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Volume 12 No. 4
October • 1957

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journal of dental medicine

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JOURNAL OF DENTAL MEDICINE

VOL. 12 OCTOBER, 1957 NO. 4

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Subscription rate: $4.00 Annually, $4.50 Foreign. Single copies, $1.00. Special Nutrition Issue (Sept. 1953) $1.00 per copy, special rate in quantity. Tenth Anniversary Issue (April 1956) $1.00 per copy, $5.00 for bound volume.

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THE "PAIN SYNDROME" OF THE TEMPOROMANDIBULAR JOINT

HUNTER L. HARANG, D.D.S., F.I.C.A.*

The diagnosis and treatment of the "pain syndrome" of neuralgia in and about the temporomandibular joint demands of the diagnostician a complete knowledge of the anatomy, physiology, neurology, pathology, and mechanical functions of such a complex joint.

Otolaryngologists, dentists, and general practitioners of medicine often encounter patients who complain of such symptoms as vertigo, various degrees of hearing impairment, eustachian tube symptoms, such as "stuffy feelings" in the ear, tinnitus, and seemingly catarrhal deafness, with atypical headaches. Other symptoms include glossodynia, burning sensations of the tongue, or abnormal dryness of the mouth. These symptoms have been described by many authors, the most familiar being the works of Costen. They are well understood and are immediately recognized.

These symptoms, evolving from dysfunction of the temporomandibular joint, are so manifested as to include many structures of the head and neck. Since some are real and some are imaginary, there is small wonder that confusion exists in the volumes of written theories proposed for the treatment and diagnosis of the similar syndromes.

It is the "pain syndrome" of the temporomandibular joint that should be diagnosed as a separate causalgia and thus treated with a definite approach. The understanding of Gerard's physiology of pain and Ruben's correct control of pain are essentials in controlling the "pain syndrome" of the temporomandibular joint.

Pain stimuli of the neural origin are generally auriculotemporal neuralgia, glossopharyngeal neuralgia, with paraesthesia of the tongue and pharynx, headaches, tic douloureux and atypical tri-facial neuralgias and neurological pains simulating those of tic douloureux.

* Diplomate, American Board of Oral Surgery; Fellow, International Anesthesia Research Society.

Pain Classified As To Origin

1. Neurological
2. Psychosomatic
3. Traumatic
   a. Extracapsular injuries to the temporomandibular joint.
   b. Intracapsular injuries to the temporomandibular joint.
   c. Rupture of the meniscus.
   d. Injuries to the perichondrium.
   e. Fractures of the bony components of the temporomandibular joint.
4. Dental origin
   a. Neuritis from dental pathology.
   b. Periostitis from dental pathology.
   c. Periosteomyelitis from dental pathology.
   d. Tumors with extension.
   e. Dental malocclusion.
   f. Cuspal interference.
5. Pathological
   a. Osteoarthritis.
   b. Polyarthritis.
6. Physiological
   a. Overdevelopment.
   b. Underdevelopment.
   c. Anomalies.

PROFESSIONAL OBLIGATIONS TO PATIENTS

Patients suffering from temporomandibular joint neuralgia usually seek advice of otolaryngologists, dentists, general physicians and neurologists. It is the professional duty of these specialists to have a universal knowledge of the neurological pain and its associated functions with this type of joint condition and to maintain a close cooperation by consultations. The patient seeking relief should have a careful clinical examination, and a careful history should be taken and recorded, possibly several different times, to reveal as much light as possible on the true background. Before a final diag-

DIFFERENTIAL DIAGNOSIS

	Paroxysmal Trigeminal Neuralgia or Tic Douloureux	*Temporomandibular Joint Neuralgia*	*Atypical Facial Neuralgia or Psychalgias*
ONSET:	Quick onset never having been present.	Pain gradual with increase of severity.	May be any form, acute or constant.
TYPE:	Short and quick.	Shooting, dull or constant.	Usually closely related to paroxysmal.
DURATION:	Short periods, 2 or 3 minutes. Leaves as fast as it comes.	Lasts for long periods.	Usually 3 minutes. May be constant.
FUNCTIONAL STIMULI:	No effect as to function. Pain may hit at any time, usually when awake.	Severe after talking or eating, may awaken patient.	None necessary.
DISTRIBUTION:	Follows definite nerve distribution. 1) mandibular branch 2) maxillary branch	No pattern. 1) dull headache 2) preauricular 3) postauricular 4) down to cervical area	Due to psychosomatic stimuli may be located anywhere.
AGE:	Usually above 45.	No respect to age—18-90.	Usually above 25.

nosis is given, the patient should be referred to the related specialists for consultation. Careful treatment and observation should be carried out in proper departments to which the patient has been referred.

Time after time it has been shown that to be correctly diagnosed and treated, those cases must have the cooperation of at least one specialist from each of the above fields, and unless the complex problem involved in the correct diagnosis and treatment is fully understood among these fields, the patient will not have a satisfactory treatment or recovery.

The history in itself reveals that many of the pathetic patients who have gone from antral washings to teeth extractions, to x-rays for brain tumors, back to psychiatrists and back around again to neurologists, could have been relieved if the specialists had cooperated to the fullest extent in the beginning. It is our responsibility therefore, as diagnosticians, to further our knowledge in this field so that we may immediately and correctly diagnose these cases and start on a conservatively constructed treatment.

ANATOMY

It is not in the scope of this paper to go into the detailed anatomy of the temporomandibular joint, with which every student should familiarize himself. The temporomandibular joint is a complex joint. Associated with its dysfunction are painful symptoms, extending over the side of the head, face and neck. Costen grouped these symptoms into the syndrome which now bears his name. Anatomists have proved conclusively that Costen's explanation of the syndrome is anatomically impossible. Sicher is one of the anatomists who proved that this theory is impossible. Harris brings out, in his anatomy of the temporomandibular articulation and ad-

jacent structures, a beautiful resume of anatomy and osteology with the influence of hypertonicity of the masticating muscles. His quotations from Dr. Monson are very important and every student of the temporomandibular joint should be familiar with his writings. However, Sicher disapproves of his concept of pain. The "pain syndrome" has not been so defined nor diagnosed as the Costen "symptoms."

Such things as anxiety neuroses should be fully understood and should be anticipated in these types of syndromes.

The acute inflammatory and traumatic conditions of the joint will respond to the conservative management which includes rest, occlusive adjustments and limitation of motion. In long standing chronic conditions with internal derangement of the temporomandibular joint, conservative management as a rule gives no relief. In long standing chronic conditions in which there is interference with function, the extirpation of the affected meniscus insures excellent results; however, before going into the differential diagnosis and treatment, it is not our purpose to indicate that all of these cases should be operated upon.

APPROACH AND BACKGROUND

Linsay's work, Transactions of British Society For The Study of Orthodontics, "Changes in the Bone of the Mandible and the Temporomandibular Joint Incident to the Application of the Orthodontic Forces," shows changes in the glenoid fossa, on the head of the condyle, in the angle of the joint and the alveolar process of the mandible. Absorption was marked by osteoclasts on the anterior part of the glenoid fossa and on the head of the condyle, this being the side of the pressure. This experiment was done for eighty-two days on Rhesus monkeys. Rhesus monkeys were used for the experiment because of the similarity of the temporomandibular joint, and the occlusion of these animals approaches most nearly that of man.

The great works of Harom Shohet, Paul Edmond, and H. L. Harris should be referred to for interesting articles on the subjects of vertical dimensions and positioning of the mandible with full denture prosthesis, partial denture prosthesis and balancing occlusion. These will not be discussed further, but the knowledge should be maintained for constant reference.

March Robinson's article, "The Temporomandibular Joint" brings out the fact that the tissue found in the joint is not of the type found in stress bearing areas; therefore, it is reasonable that there should be no appreciable stress in the temporomandibular joint. Joint function as stressed, will be explained by a direction of muscle action. The principle of the neuromuscular regulations of the joint itself is presented by Robinson as a theory of reflex control. One important point he brings out is that the joint is lined by synovial membrane and the articular disc is not composed of fibrocartilage which is not repairable, but is a specialized connective tissue which is capable of repair. In no area of the body is a synovial layer to be covering a stress bearing area of a joint. Repair of lesions of the disc can occur if the condition responsible for the pathological process is corrected.

It is well for us to comprehend that Goodfriend has termed the course of the disease factor. Time, effort, and money may well be expended unnecessarily if the patient seeks advice of those physicians and dentists who do not fully understand the implications of these temporomandibular joint dyscrasias.

It should be brought out that it also takes the participation and the cooperation of the patient in all of these cases. He must be concerned enough with the care of his own case in order to seek treatment and to follow through with the procedure outlined for his treatment.

THE PATIENT IN PAIN

Let us take the case as it is presented: We quote Dr. Meyer Schier here:

"How do we approach the problem of reorientation of mandibulomaxillary relations? Every effort is made to disengage accustomed joint function or dysfunction. In sequence, as may be indicated, high cuspal irregularities are reduced

to permit ease of movement in all directions on the horizontal plane, and supplemented and implemented by splints with neointerdigitive occlusal planes. The fundamental endeavor is directed to an attempt at release of the believed to be abnormal nerve, muscular and vascular status of the joint and adnexa due to trauma, to a state of free mandibular mobility devoid of previous conscious or unconscious dysfunction or traumatic habit." This is our aim when the patient presents us with distress and pain.

THE CASE HISTORY OF MRS. W. T.

The patient, age 35, seen in acute distress with extreme pain, inability to eat, masticate or carry on a normal functional life due to inability to talk. The history revealed that this patient had visited twenty-two doctors in a period of eleven years.

She had letters from neurologists and neurosurgeons from outstanding clinics in the country stating that she presented a case of "a typical facial neuralgia" for which there was no cure, and they advised her not to seek any further medical advice due to expending her finances.

A careful history revealed that this patient, at the age of approximately twenty-two, suffered an infection of a lower left third molar, which was removed. Evidently she developed a "dry socket" with excruciating pain. After seeking treatment from several different professional men, she thought it was another tooth hurting and "talked" a dentist into removing the second molar. As a result the pain was more exaggerated. Before that particular tooth was completely recovered, a dentist took out the upper second molar, hoping that would relieve her pain. I am sure this pain was at first a typical type of dry socket with radiating pain into the side of the face and to the ear. The patient then went three to four years before developing some type of syndrome in this left temporomandibular joint. She visited another dentist and had the rest of the molars removed from the left side of the maxilla and mandible. Due to her extreme overbite, all of her mastication had to be done on the right side to obtain any type of functional occlusion. This patient began to have headaches, atypical in type, of the type usually found in temporomandibular dyscrasias. The pain could not be localized. It was not originating from the ear, and was not a preauricular or postauricular type of pain. The patient visited numerous physicians with headaches and it was the same old story. She went from one physician to the next, first on sedatives of a weaker variety, increasing the dosage from one physican to the next. Several internists saw her. X-rays finally were taken of the head for brain tumors. Neurologists saw this patient and finally a neurosurgeon was called in. His diagnosis was tic douloureux, and an alcohol block of the second division of the fifth nerve was attempted. It was not successful and in a few months was repeated. This time the alcohol block was successful but yet the pain persisted. After many visits to the neurosurgeon, she was sent to a psychiatrist. After the psychiatrist talked with her several times, he recommended another neurosurgeon. The patient was passed from one physician to the next.

This patient had the continuous type of headache over a span of ten years, so that now it was becoming quite a psychosomatic type of problem. This lengthy history was taken by tape recorder and at the conclusion of the history, the diagnosis was made of a combination of psychosomatic pain with actual temporomandibular join pain with an overlaying factor of muscle spasm and vascular spasm of the vessels throughout the entire distribution of the carotid artery on the left side of the face. At the same time, an appointment was made with the general dentist for impressions and the construction of a bite plate. In four days, the insertion of the occlusal rest, or bite opener, was inserted. In the meantime, I had referred this patient to a neurosurgeon, who substantiated our therapy with stellate blocks to try to relieve the tremendous muscle and vascular spasm of the left side of the face due to the previous pain and the alcohol block injections. These stellate ganglion blocks result were recorded with technicolor movie films showing the actual block in process. The patient wore this appliance for ten days, after which she returned completely symptom free for the first time in ten to twelve years.

CASE HISTORY OF E. W. AGE 46

This patient was seen in acute pain, referred by an EENT physician. This patient came to us completely "worked up." As far as history, x-rays, etc., EENT Consultation for infection of the antrum, mastoids, middle ear, otosclerosis, nasal and pharyngeal infection was negative.

His dental occlusion and his dental condition was readily observed to be completely normal. He had prefect occlusion of all his teeth. He had intercuspal relation. He had movements protrusively in any direction, no overbite or malposition or malocclusion.

His past history revealed that this man was a Golden Gloves champion boxer in his weight. He was a Marine flyer and was in several explosions in airplanes, had been shot down, and had received tremendous trauma to his mandible.

The onset of pain occurred two weeks previously while eating a steak dinner. The pain was so intense and so terrific and with such impact, that it completely blacked him out enough to knock him to the floor. He had to be revived from an unconscious state. His pain was so great from then on until the time of operation, that he could not eat. He presented himself to our office in this extremely painful condition. He had been taking quite a bit of sedatives for relief of pain and actually it did not stop his pain, it only allowed him to live with it.

After diagnosis was made, we put him in the treatment room and gave him a direct injection of Xylocaine into the affected joint. He had immediate relief for the first time in two weeks, and could open his mouth. He went out and ate a big meal consisting of steak, and within two hours after the injection had worn off, his pain returned. This man came to surgery in about two or three days and we did a menisectomy under local anesthesia which was recorded in colored movies. The patient was placed back in bed and that night when rounds were made in the hospital, he was found eating his supper which included scrambled eggs and ham. He had been completely free of any painful symptoms and the time elapsed has now been over two and one half years.

Moving pictures were recorded of his protrusive movements, his lateral excursions of his mandible three months postoperatively. He had perfect excursion, both protrusive and lateral. His opening of his jaw is normal. There is no impairment or impingment or impairment of function in his articular movements.

OPERATION

The patient is prepared for surgery and medication is given. Under local anesthesia an incision was made starting in the fold at the anterior margin of the helix at its upper attachment. The incision is carried downward in the fold to the upper portion of the tragus, and extending down to the anterior fold of the lobe to the lower attachment of the lobe. The incision is carried through the skin and subcutaneous tissues, exposing the cartilage of the tragus, down to the masseteric fascia. The tissue is dissected by blunt dissection, anteriorly for about 2 cm. The anterior branches of the temporal vessels are cut, ligated, and retracted forward with the superior pole of the parotid gland. The joint is now exposed. An incision is made through the fascia in a circular manner, bringing the tail of the incision down toward the neck of the condyle. The joint is entered by means of a transverse incision, paralleling the border of the meniscus into the upper compartment of the joint, and another into the lower compartment of the joint. The lateral margin of the meniscus is then exposed and is grasped with a hemostat. The disk is then dissected from its anterior, posterior and medial attachments. The cavity is carefully freed of any loose fragments, the capsule is closed with 4-0 gut sutures. The fascia and subcutaneous tissues are closed with 4-0 gut sutures. The skin edges are now closed with 4-0 atraumatic dermalon sutures. The wound is dressed with a pressure dressing. The patient is not instructed to limit motion. A soft diet is maintained for several days.

SUMMARY

May we summarize here by saying that the purpose of this paper is to bring out the drastic

need of correct diagnosis and the correction of pain in the temporomandibular joint neuralgia. It is our desire to show that these dysfunctions are atypical and each new patient presents a little different problem in diagnosis and treatment.

The diagnosis and treatment of the temporomandibular joint is a responsibility for the diagnosticians of all allied fields. The diagnosis of neuralgia from a dysfunction of the temporomandibular joint can only be made after a careful history and clinical examination.

It is very pointedly brought forth that one should be familiar with the writing of: Costen, Harris, Monson, Sicher, Schier, Shohet, Paul Edmand and Robinson. Anderson, in his article, has brought out the need for otolaryngologists, dentists, and general practitioners to avail themselves of the new and present concepts of neuralgias resulting from dysfunction of the temporomandibular joint.

Care should be exercised in handling these patients, for it must be remembered that they have usually consulted many specialists and have developed a "neurosis" along with the pain of their dysfunction. Therefore, conservative methods of treatment must be applied. These patients will have to be treated with gentle manipulation of instruments and a deep understanding of the "occlusal balance," bite opening and bite raising, etc. Palliative and conservative treatment should be carried out first, such as immobilization, rest, soft diet, application of dry heat, intra-articular dosages of Hydrocortone and administrations of muscle relaxant and tension relaxant drugs. The results of conservative treatment are very satisfactory with 95% of the cases responding and being relieved of pain. Cases with long standing illness should have correct repositioning of the mandible and bite opening and balance of the occlusion for the relief of pain. Intracapsular injection of Hydrocortone, along with the oral administration of balanced cortone therapy will be helpful to those menisci that may have many degenerative changes.

By conservative treatments, 95% of the painful and acute temporomandibular joint neuralgias will be relieved. It is only necessary to do a radical surgical procedure (menisectomy) when the meniscus has been ruptured by trauma or the damage to the structures are irreversible.

Therefore the conservative methods must be applied first. These patients should go to dentists who will be gentle in manipulation of instruments and who will treat the psychalgias met with, along with the true tic douloureux and temporomandibular joint neuralgias. The diagnosis and treatment of these neuralgias is the responsibility of all allied fields. Palliative and conservative treatment should be carried out first. The results are very satisfactory with these cases responding to relief of pain. Only those cases that have been present for long periods of time and have caused a large amount of dysfunction with damage that is irreversible, will have to be operated by menisectomy. Successful treatment of the pain syndrome of the temporomandibular joint depends upon differential diagnosis and subsequent subjective treatments.

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Houston, Texas

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LE "SYNDROME DE DOLOR" IN LE ARTICULATION TEMPOROMANDIBULAR

Dr. chir. dent. Hunter L. Harang (F.I.C.A.)

SUMMARIO IN INTERLINGUA

Le objectivo del presente articulo es signalar le urgentia del correcte diagnose del dolores in casos de neuralgia del articulation temporomandibular. Omne caso individual de iste dysfunction es "atypic," e omne nove patiente presenta in un certe mesura un nove problema diagnostic e therapeutic.

Le diagnose e therapia de affectiones del articulation temporomandibular es le responsabilitate del diagnosticistas de omne le campos implicate. Le diagnose de neuralgia como resultato de dysfunction del articulation temporomandibular es possibile solmente super le base de un precise historia e un exacte examine clinic.

Le preoccupation con patientes de iste genere require alte grados de circumspection, proque on non debe oblidar que illes ha generalmente consultate multe specialistas e que a parte le dolor de lor dysfunction illes ha disveloppate un specie de "neurosis." Ergo, methodos conservatori debe esser usate in lor tractamento. Iste patientes debe esser tractate con le plus caute manipulation possibile del instrumentos e super le base de un profunde comprension del "balancia occlusional" e del factores implicate in illo. Al initio, un tractamento conservatori e palliative debe esser usate. Isto include mesuras como immobilisation, reposo, dieta molle, application de calor sic, dosages de hydrocortona intra-articular, e administrationes de drogas que relaxa le musculos e le tension general. Le resultatos de un tal tractamento conservatori es multo satisfactori. In 95% del casos, alleviamento del dolores e un responsa generalmente positive es obtenibile. In casos de dysfunction a longe durantia, il es necessari effectuar un correcte repositionamento mandibular e balancia occlusional pro alleviar le dolores. Injectiones intracapsular de hydrocortona, insimul con administration oral de un balanciate therapia a cortona, va esser utile in casos in que le meniscos ha suffrite alterationes degenerative.

Le tractamento conservatori pote alleviar le dolorose e acute neuralgias del articulation temporomandibular in 95% del casos. Radical intervention chirurgic (meniscectomia) es necessari quando le menisco ha essite rupturate per trauma o quando le lesiones structural es irreversibile.

THE INTRADERMAL ASCORBIC ACID TEST PART I. A REVIEW OF ANIMAL STUDIES*

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INTRODUCTION

It has been long recognized that the disease state cannot be described in pure blacks and whites. In other words, the proof is clear that health and disease appear *clinically* in shades of gray.[1] True enough, in some instances the patient presents himself with classical symptoms and signs of a specific disease process. In such a case, the diagnosis is readily apparent. But, in a greater percentage of instances, the individual, though not *clinically* ill, is nonetheless *biochemically* disturbed. The problem becomes one of ascertaining the presence and finally the extent of the disability. Toward this end, the investigator utilizes the examination of blood, urine, feces, saliva and similar media. It is presumed that an analysis of one of these materials will aid in establishing the state of health. But here again, one is often making judgments on the basis of blacks and whites. Often a so-called normal or marginal hemogram, for example, is regarded as good evidence of the absence of anemia. In the final analysis, when an individual is biochemically diseased, the derangement is in the hinterland, as it were, in the *tissues*.

A hemogram, a urinalysis, a measure of something in the cerebrospinal fluid presume that cell changes reflect immediately in the blood and/or urine and/or spinal fluid. The concept is now coming to the fore that cellular disease can and does occur long before there is clinical or even laboratory proof of pathosis in the blood, urine, and feces.[2, 3, 4, 5]

It is quite understandable, therefore, why attempts are now being made to study the health status of the individual cell and its aggregates. Evidence that much valuable data can be so derived is underscored in the following illustration:

It is generally known among clinicians that a urinary sugar level is a significant index of diabetes mellitus. However, it is also quite clear that a fasting blood sugar will often disclose the diabetic state even when the urine is sugar-free. Moreover, authorities agree that a glucose tolerance test is a more sensitive index of diabetes mellitus than either a fasting blood glucose or a glucose-urine determination. However, not often is it recognized that the glucose concentration of the tissues themselves may be an even more delicate barometer than the blood and urine procedures. This concept, namely that of seeking diagnostic evidence of disease at the cellular level, is shown quite dramatically in the following case report:

A forty-nine year old male had been suffering five years from severe, constantly recurring furunculosis. During this period of time, he had been obliged to seek hospital treatment ten times. In view of his obesity, blood glucose tolerance tests were repeatedly done. Since they repeatedly showed only normal limits, it was impossible to make a diagnosis of diabetes mellitus. An examination of the skin sugar concentration showed a fasting level of 81 mgm. percent (normal 55-68 mgm. percent). The patient was placed on a strict diabetic diet and given 10 units of insulin thrice daily. By

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the end of the eighth day, the furuncles had vanished completely. Thereupon, by way of an experiment, the patient's carbohydrate intake was returned to normal levels. This had to be stopped on the third day because of the reappearance of the furuncles. Strict adherence to a diabetic diet plus 30 units of insulin daily again served to clear up the condition and the patient suffered no further attacks for seven days. Once again, the regular carbohydrate diet was re-instituted and again it had to be discontinued after four days because of the recurrent furunculosis.[6]

It is not our purpose here to discuss the possibility of cutaneous diabetes mellitus. Suffice it to say that only through a study of the tissues was it possible to reach a satisfactory solution to this patient's problem.

Essentially the same problem prevails in connection with the ascorbic acid status of the individual. The question constantly recurs as to the significance of the plasma ascorbic acid level, the urinary excretion, and the concentration of ascorbic acid in the leucocytes. In an attempt to reach the tissues, an intradermal decolorization test has been developed. It is our purpose in this report to review the experience with this test in laboratory animals. In subsequent publications we shall consider human studies[7] and analyze a series of our own patients.[8]

REVIEW OF THE LITERATURE

Attempts have been made during the last two decades to evaluate an intradermal ascorbic acid test in both (1) animals, and (2) human beings.

ANIMAL STUDIES

Just two decades ago, Rotter[9, 10] recognized the need for a rapid clinical test to estimate the body concentration of ascorbic acid. It occurred to him, while measuring urinary ascorbic acid excretion by titration with 2,6 dichlorophenolindophenol, that the time for decolorization of an intradermal injection of this same dye might provide an index of tissue ascorbic acid concentration. In his initial experiments, he injected a "small quantity" of a N/1000 sterile solution of 2,6 dichlorophenolindophenol into the soles of control and scorbutic guinea pigs. It was his opinion that the dye was decolorized much more rapidly in the control group as compared with the scorbutic animals. In order to eliminate the possibility of dye absorption, he simultaneously injected methylene blue intradermally. It was clearly shown that the dye is truly decolorized, not absorbed, since the methylene blue remained clinically unchanged after the DCPIP* had vanished. Furthermore, extremities cut off from circulation revealed time scores similar to those of normal limbs. Thus, Rotter concluded the phenomenon to be a chemical reduction.

Gambigliani-Zoccoli and Lombardo,[11] employing Rotter's technique, carried out a series of investigations. In the first study five pigs were placed on a scorbutic diet until classical scurvey became apparent. The original decolorization times of two to four minutes remained the same during this entire scorbutic period. A second study of "some" normal animals was then instituted, wherein each guinea pig received a parenteral injection of 50 mgm. of vitamin C. Intradermal decolorization times were determined before, one hour later and 24 hours after the injection. The original decolorization times averaged two to four minutes. One hour after injection they had diminished to 30 seconds. The twenty-four hour scores remained reduced with an average of 60 seconds. No specific scores were listed for each animal in any of the studies. In a third study scorbutic animals were given the same 50 mgm load dose. Pre- and post-injection times were precisely the same. Gambigliani-Zoccoli and Lombardo felt, on the basis of these observations, that the Rotter intradermal test was not a measure of ascorbic acid status. They justify this conclusion in the following fashion. The scorbutic animal needs ascorbic acid. When it is injected, the vitamin is promptly taken up in all of the tissues. Consequently, little of it reaches the skin. Therefore, the intradermal time remains unchanged. In contrast, the control animal is already saturated. Thus, the injection of the

* Hereafter, DCPIP will be used as the abbreviation for 2,6 dichloro-phenolindophenol.

vitamin results in its free circulation in the blood and the intradermal time decreases. They further objected to the intradermal test on the grounds that it is not specific for ascorbic acid since the dye is decolorized by other reducing agents. Gambigliani-Zoccoli and Lombardo did, however, suggest that a comparison of the intradermal time before and after injection of a load dose may provide significant information about the ascorbic acid status.

The next year, Banerjee and Guha[12] studied a group of twenty guinea pigs. They employed a N/400 concentration of the dye and produced a 2mm. wheal in the shaved abdomen. It should be emphasized that, in animal investigations, Banerjee and Guha differed from Rotter who used an unknown quantity of N/1000 test dye. The animals in this study were first made scorbutic and then returned to normal health. This was accomplished by the daily dietary administration of 4 to 8 mgm. of ascorbic acid to the scorbutic animals. These investigators noted that the decolorization time was inversely related to the changing weight of the guinea pigs. In other words, as the animals lost weight (as they do on a scorbutic regime), the decolorization time increased at the same rate. Three other groups, each composed of five animals, were used for another experiment. All animals were fed scorbutic diets. The first group received a supplement of 2 mgm. of ascorbic acid daily, the second 1 mgm. and the third group 0.5 mgm. The mean decolorization time was shortest in the first group (those receiving the greatest vitamin supplement). There was no appreciable difference in the decolorization time in those pigs receiving 1 versus 0.5 mgm. ascorbic acid daily.

Suzuki[13] intradermally injected varying amounts of DCPIP into the earlobes and lateral abdominal walls of two rabbits and one guinea pig. The times varied significantly between the two sites. They were prolonged as greater quantities of solution were employed. For example, in one rabbit, injections of .01, .02 and .03 cc. of dye into the earlobe yielded times of 3.5, 5 and 8 minutes respectively. Consistently longer decolorization times were noted in the guinea pig as opposed to the rabbits. Suzuki felt that this was a manifestation of the guinea pig's inability to synthesize and store the vitamin.

Cera[14] was interested in verifying the results of Gambigliani-Zoccoli and Lombardo.[11] He employed Rotter's technique in guinea pigs. Five wheals were produced on each animal and the average decolorization time was calculated. He first performed the test on six normal guinea pigs and found a group time range from 3½ to 5 minutes. These same six animals were then placed on a scorbutogenic diet. On the twenty-first day, when scurvy was obvious, the test time was repeated. The scores obtained were almost exactly what they had been under normal dietary conditions. A second group of six pigs was allowed to become scorbutic. They were then administered 50 mgm. of ascorbic acid intraperitoneally. One hour later, the skin times were repeated. Only an insignificant reduction of ½ to 1 minute occurred in four cases. However, a third aggregate of six normal animals was subjected to the same 50 mgm. dosage. This last category was tested before, 1 hour later, and 24 hours after the vitamin C injection. The reduction in time ranged from 67 to 87 percent in one hour. Even after 24 hours the diminution ranged from 50 to 75 percent of the original value. Cera tried to explain the apparent paradox by suggesting that, in scorbutic animals, the injected ascorbic acid never reaches the skin. The implication is that the skin enjoys a lower priority than other organs and thus is by-passed. Secondly, he speculated that the injection traumatically alters the usual dermal status, rendering the decolorization time a poor measure of the actual skin conditions.

Rapaport and Miller,[15] employing a N/400 dye solution, raised four 2 mm. wheals simultaneously on the shaved abdomens of healthy rabbits. The reagent decolorized in 5 minutes or less. These researchers observed that only insignificant differences in decolorization time were obtained when the four intradermal injections were made simultaneously. Methylene blue administered at the same time remained unchanged for at least 24 hours. From these observations they felt that, not only was an

absorptive phenomenon for the ascorbic acid dye eliminated, but also that this test was a fair index of the vitamin C status.

Masuzawa,[16] using Rotter's technique, maintained six and three guinea pigs on scorbutic and normal diets respectively for twenty-one days. Skin tests were made in the soles every three or four days and the weights recorded at that time. During the twenty-one days, the decolorization time in the scorbutic animals increased from an average of 5.7 minutes to 49.5 minutes. The test time in the animals on the normal diet declined from 7.3 to 1.7 minutes. The average weight loss in the scorbutic group was 27.2 percent. The normal group gained in weight an average of 18.1 percent. The author concluded that the decolorization time parallels tissue saturation with vitamin C, even though he recognized that other reducing substances such as glutathione and cysteine were present. He concluded that times of 4 minutes or less equal saturation; 5-10 minutes normal; and over 10 minutes signifies a deficiency state.

Banerjee and Guha[17] felt that a saturation test could be developed using the skin test time. They believed that a necessary prerequisite to such a study would be the establishment of a minimal decolorization time. This would provide a reference point for evaluating decolorization time alterations following load dosing. To this end, they employed guinea pigs and human beings in a saturation study. Thirteen guinea pigs were divided into three groups according to the ascorbic acid supplement. Group #1 (7 animals) received 15 mgm. ascorbic acid daily for 14 days, 40 mgm. for the 15th and 16th days, and 100 mgm. on the 17th, 18th and 19th days. The intradermal test (technique of Rotter) was performed on the shaved abdomen every 2 to 4 days. The initial skin times ranged from 1.6 to 3.1 minutes with an average of 2.5 minutes. On the 9th day, the decolorization times averaged 1.7 minutes. By the 17th day all the animals yielded decolorization times of 90 $\pm$ 5 seconds. Additional ascorbic acid for another two weeks did not further decrease the time. Apparently 90 seconds represented complete saturation. Two other groups of animals (3 in each category) were supplemented with 2 and 4 mgm. daily dosages of ascorbic acid. These animals were maintained for 29 days at this supplement level. As before, the skin time was performed every 2 to 4 days. The second group (2 mgm. supplement) exhibited an initial time of 2.5 minutes. Twenty-eight days later, the times ranged from 1.5 to 1.8 minutes with an average of 1.6 minutes. The third group (4 mgm. supplement) declined from a 3 minute average (range 2.2 to 4.5 minutes) initially to a score of 1.5 minutes on the 28th day. With the lower supplementation of ascorbic acid (2-4 mgm. as opposed to 15 mgm. per day), four weeks were required to reach a saturation score. On the other hand, the heavily supplemented animals (15 mgm. per day) attained a minimum time of 90 seconds in about two weeks.

In a third publication, Banerjee and Guha[18] reported on the plasma and urinary ascorbic acid levels and the intradermal decolorization times of two groups of animals. One group of 17 guinea pigs and another of 16 were maintained for three weeks on normal and scorbutic diets respectively. At the close of the experiment, the ranges of the intradermal times for the two groups were 1.5 to 4.7 (normal group) and 5 to 15 minutes (scorbutic group). It is clear from these figures that the values for the normal group were much lower. However, the authors emphasized that, given a specific plasma ascorbic acid value, it was impossible to predict the decolorization time.

The same investigators[19] placed three groups, each of 12 animals, on different diets: (1) scorbutic, (2) normal, and (3) normal diet plus a daily injection of 100 mgm. of ascorbic acid. On the day before sacrifice, approximately three weeks later, intradermal times were determined. In addition, ground liver specimens were analyzed for ascorbic acid content. These investigators found that the minimal decolorization time (90 seconds) was observed in the group which received supplemental vitamin therapy and the maximal test time (4.5-9.2 minutes) in the scorbutic group. The greatest liver concentration (18.2-31.3 mgm. percent) was found in the group receiving supplementation. The authors concluded that the time necessary

for reducing the dye was lowest when the liver was saturated. This they felt established the skin time as a fair index of body saturation with ascorbic acid.

From earlier studies, Banerjee and Guha had noted that the conjugated forms of urinary ascorbic acid diminished in parallel with the increase in body saturation. Thus, they speculated that the disappearance of these combined forms from the urine might represent the limit of saturation. In their last publication, Banerjee and Guha[20] injected 100 mgm. of ascorbic acid into each of 14 healthy guinea pigs. It was found that the conjugated forms of urinary ascorbic acid disappeared under these conditions. Concurrent intradermal times, however, exceeded 90 seconds. This is of interest since 90 seconds was regarded as the intradermal index of saturation. Thus the animals were considered to be unsaturated at the time when the combined forms of ascorbic acid vanished from the urine.

Slobody[21], in seeking a more optimal dye concentration and wheal size, utilized the shaved abdomens of three rabbits in a pilot study. He injected varying quantities and concentrations of the dye intradermally. He found that a wheal size of 4 mm. and a DCPIP solution strength of N/300 yielded greater score constancy in any one animal. He felt that the employment of a more concentrated dye and a larger wheal minimized the effects of other reducing agents in the skin. The time taken for decolorization was lengthened and thus easier to read.

Curatola[22] employed four groups of guinea pigs in evaluating Rotter's test. The first group consisted of 10 animals which were fed a liberal diet for 15 days. After 8 hours of fasting, a .01 cc. portion of a N/400 solution of DCPIP was injected into the sole of each posterior foot. The next day the injections were repeated with .03 cc. of a N/1000 concentration of the dye. Little difference was noted between the decolorization times of the N/400 and N/1000 solutions. Consequently, he utilized .03 cc. of the N/1000 dye throughout the rest of the studies. Group 2 (6 guinea pigs from group 1) was placed on a 15 day deficient diet and then sacrificed to determine the ascorbic acid of the following organs in mgm./100 grams of tissue: (1) suprarenal, 0.85 $\pm$ 0.06, (2) liver, 0.25 $\pm$ 0.07, and (3) testicle, 0.18 $\pm$ 0.05. Ten animals, comprising group 3, were placed on a scorbutogenic diet for 15 days. At the beginning and end of the period, the serum ascorbic acid was determined. Every three days, the skin time was taken in the plantar area of both hind feet. At the close of the experiment the animals were sacrificed and the vitamin C estimated in the same three organs. It was found that the serum ascorbic acid level declined from .13 to .07 mgm. percent. The decolorization time rose from 3 to 4.4 minutes. Group 4 consisted of four animals which had received a vitamin C rich diet for 15 days. The intradermal times at the end of the period were very low—between 1 and 1.5 minutes. The guinea pigs were then given 80 mgm. of ascorbic acid subcutaneously. The disappearance times were measured after 10 minutes and then every hour for 7 hours. The times decreased in value very slowly, with the lowest score at the sixth hour. The maximal decline varied from 30 to 45 seconds. A scorbutic diet was then instituted and continued for 14 to 17 days. The skin times were performed every 3 to 4 days. They rose from 1-2 to 5-7 minutes. At the end of this period another 80 mgm. portion of ascorbic acid was injected into each animal. As before, the intradermal times were determined after 10 minutes and each hour thereafter for 7 hours. After 10 minutes they had declined from 60 to 80 percent. The lowest times were reached at the third hour, after which they began to rise again. He concluded that the test is a crude index of ascorbic acid status of the skin—the cutaneous level is not necessarily indicative of body saturation. Times of $\frac{1}{2}$ to 3 minutes signified an adequate skin concentration. Intermediate scores (3 to 5 minutes) required clarification by load-dosing. Times in excess of 5 minutes could be regarded as indicative of poor cutaneous saturation. Intermediate scores which necessitated load-testing were interpreted as follows: (1) normal subjects exhibited a slow decrease in decolorization time reaching a low point at the sixth hour, and (2) deficient subjects revealed a more accel-

TABLE 1

Author(s)	*Amount of Injected Dye (cc.)*	*Wheal Size (mm.)*	*Injection Site*	*Concentration of Dye*
Rotter (9)	?	?	guinea pig soles	N/1000
Rotter (10)	.01	2	guinea pig soles	N/400
Gambigliani-Zoccoli and Lombardo (11)	.01	2	guinea pig soles	N/400
Banerjee and Guha (12)	.01	2	guinea pig, shaved abdomen	N/400
Suzuki (13)	.01 .02 .03	2	2 rabbits, earlobe, lateral abdominal wall	N/400
Suzuki (13)	.01	2	1 guinea pig, earlobe and lateral abdominal wall	N/400
Cera (14)	.01	2	guinea pig soles	N/400
Rapaport and Miller (15)	?	2	rabbit, shaved abdomen	N/400
Masuzawa (16)	.01	?	guinea pig sole	N/400
Banerjee and Guha (17)	.01	2	guinea pigs, shaved abdomen	N/400
Banerjee and Guha (18)	.01	2	guinea pigs, shaved abdomen	N/400
Banerjee and Guha (19)	.01	2	guinea pigs, shaved abdomen	N/400
Banerjee and Guha (20)	.01	2	guinea pigs, shaved abdomen	N/400
Slobody (21)	.05	4	rabbits, shaved abdomen	N/500 N/1000 N/2500 N/5000
Curatola (22)	.03	?	guinea pig sole	N/1000

erated descent and a maximal decrease at the third hour. It is of parenthetic interest that Curatola found no relationship between the amount of vitamin C in the organs and Rotter's time.

SUMMARY

From a review of the literature it is clear that all available data is based on the use of 2, 6 dichlorophenolindophenol dye. Table 1 reveals that: (1) in every study but three the dye concentration was N/400, (2) in all but three instances guinea pigs were employed as the experimental animal, (3) when wheal size is reported, it has been with one exception 2 mm. in diameter, and (4) the quantity of test dye has, in all but three instances, been 0.01 cc.

Table 2 summarizes the various experiments, the number and health of animals, and the duration of the investigation.

An examination of Table 3 discloses: (1) in only five of the fourteen reports is there the opportunity to compare the decolorization times at the start and conclusion of an experiment, (2) the intradermal test time is generally longer in scorbutic than in healthy animals, and (3) the administration of ascorbic acid does reduce the intradermal decolorization time according to most investigators. It is of particular note that, throughout these studies, emphasis has been placed upon the *average* measurement for the group rather than the *individual* variations during the course of the experiment.

The purposes of the animal studies, as depicted in Table 4, may be summarized as follows: (1) to establish, if at all possible, a correlation between the intradermal decolorization test time and the concentration of the vitamin in the tissues, (2) to ascertain changes in intradermal time with vitamin supplementation, (3)

TABLE 3

Author(s)	*Number and Health of Animals*	*Intradermal Time (Minutes)* *Initial*	*Final*	*Mode of Vitamin Administration*
Rotter (9)	? (N)*	?	?	normal diet
	? (S)*	?	?	scorbutic diet
Rotter (10)	? (N)	?	?	normal diet
	? (S)	?	?	scorbutic diet
Gambigliani-Zoccoli and Lombardo (11)	5 (S)	2-4	2-4	scorbutic diet
	? (N)	2-4	0.5	50 mgm. subcutaneously
	? (S)	2-4	2-4	50 mgm. subcutaneously
Banerjee and Guha (12)	20 (NS)	?	?	normal diet followed by scorbutic diet in same animals
	5 (S)	?	mean 1.8	2 mgm.
	5 (S)	?	mean 2.2	1 mgm.
	5 (S)	?	mean 2.3	0.5 mgm.
Suzuki (13)	3 (N)		.01 cc, rabbits, earlobe 3.5	None
			.01 cc, rabbits, lateral abdominal area 5.6	
			.02 cc, rabbits, earlobe 5	
			.03 cc, rabbit, lateral abdominal area 8	
			.01 cc, guinea pig, earlobe 2.5	
			.01 cc, guinea pig, lateral abdominal wall — white hair 11; brown 12; black 4.	
Cera (14)	6 (S)	3.5-5	3.5-5	scorbutic diet
	6 (S)	3.5-5	3.5-5	50 mgm. intraperitoneally
	6 (N)	?	67 to 87 percent reduction	50 mgm. intraperitoneally
Rapaport and Miller (15)	? (N)	less than 5 min.	?	normal diet
Masuzawa (16)	3 (N)	mean 7.3	mean 1.7	none
	6 (S)	mean 5.7	mean 49.5	
Banerjee and Guha (17)	7 (N)	1.6-3.1(2.5)	1.5	15 mgm. dietary
	3 (N)	2.5	1.5-1.8(1.6)	2 mgm. dietary
	3 (N)	2.2-4.5(3)	1.5	4 mgm. dietary
Benerjee and Guha (18)	17 (N)	?	range 1.5-4.7	normal diet
	16 (S)	?	range 5-15	scorbutic diet
Banerjee and Guha (19)	12 (S)	?	range 4.5-9.2	none
	12 (N)	?	range 2.5-6.5	none
	12 (N)	?	1.5	100 mgm. injected
Banerjee and Guha (20)	14 (N)	?	range 1.9-3.2	100 mgm. injected
Slobody (21)	3 (N)	N/500	(1) mean 10.6	none
		N/1000	(2) mean 5.0	
		N/2500	(3) mean 3.0	
		N/5000	(4) mean 1.6	
Curatola (22)	10 (S)	3 (average)	4.4	scorbutic diet
		?	1-1.5	rich diet
		1-1.5	5-7	scorbutic diet

*N = normal; S = scorbutic

TABLE 2

Author(s)	*Number and Health of Animals*	*Duration of Experiment in Days*
Rotter (9)	? (N)*	?
	? (S)*	?
Rotter (10)	? (N)	?
	? (S)	?
Gambigliani-Zoccoli and Lombardo (11)	5 (S)	?
	? (N)	1 hour
	? (S)	1 hour
Banerjee and Guha (12)	20 (NS)	28-30
	5 (S)	30
	5 (S)	30
	5 (S)	30
Suzuki (13)	3 (N)	?
Cera (14)	6 (S)	21
	6 (S)	1 hour
	6 (N)	1 hour
Rapaport and Miller (15)	? (N)	?
Masuzawa (16)	3 (N)	21
	6 (S)	21
Banerjee and Guha (17)	7 (N)	20
	3 (N)	29
	3 (N)	29
Banerjee and Guha (18)	17 (N)	21
	16 (S)	21
Banerjee and Guha (19)	12 (S)	21
	12 (N)	22
	12 (N)	17
Banerjee and Guha (20)	14 (N)	1
Slobody (21)	3 (N)	?
Curatola (22)	10 (S)	15
	4 (Sat)*	15
	4 (S)	14-17

*N = normal; S = scorbutic; Sat = saturated

TABLE 4

Author(s)	*Purpose of Study*
Rotter (9, 10)	To determine whether there is a correlation between the ascorbic acid tissue status and the intradermal disappearance of DCPIP
Gambigliani-Zoccoli and Lombardo (11)	To evaluate skin test
Banerjee and Guha (12)	To relate intradermal time to ascorbutic weight loss and to quantity of ascorbic acid supplementation
Suzuki (13)	To compare test times performed at two different sites with different dye concentrations
Cera (14)	To evaluate Rotter test
Rapaport and Miller (15)	To demonstrate that the dye is truly decolorized and to check the constancy of decolorization time
Masuzawa (16)	To evaluate skin test time against weight change as evidence of vitamin status
Banerjee and Guha (17)	To determine whether a minimal decolorization time is attainable
Banerjee and Guha (18)	To compare urine and plasma ascorbic acid levels with intradermal time
Banerjee and Guha (19)	To relate skin test time to liver ascorbic acid level
Banerjee and Guha (20)	To determine if disappearance of combined forms of urinary ascorbic acid indicates body saturation
Slobody (21)	To discover an optimal wheal size and dye concentration
Curatola (22)	To evaluate the skin test

to prove, if possible, that the DCPIP decolorization reflects a neutralization of the test solution, rather than just a simple phenomenon of absorption, (4) to correlate urine and blood ascorbic acid levels with decolorization time, (5) to compare liver to vitamin C levels with skin test time, and (6) to evaluate body ascorbic acid saturation with the disappearance of combined ascorbic acid forms in the urine.

The evidence (Table 5) seems to suggest: (1) a definite relationship between tissue level and intradermal time, (2) that the skin test time can be reduced with ascorbic acid supplementation, (3) that DCPIP is decolorized rather than simply absorbed, (4) that normal and scorbutic groups differ in urinary ascorbic acid excretion, blood level and intradermal time, (5) that maximal liver saturation correlates with minimal test time, and (6) lack of correlation between disappearance of combined forms of ascorbic acid in the urine and tissue saturation.

TABLE 5

Author(s)	*Conclusions*	*Basis for Conclusions*
Rotter (9, 10)	DCPIP is decolorized in the skin at a rate consistent with the amount of the vitamin present in or administered to the organism.	Scorbutic subjects showed prolonged decolorization times. Supplementation resulted in a decrease in disappearance time.
Gambigliani-Zoccoli and Lombardo (11)	Skin test is not practical for clinical use.	Scorbutic animals yielded the same skin times as normal ones.
Banerjee and Guha (12)	Intradermal time correlates with scorbutic status on the basis of weight loss and daily vitamin C consumption.	Statistical analysis indicated significant time differences in normal versus abnormal groups.
Suzuki (13)	The more concentrated solutions produce longer intradermal decolorization time. Rabbits, unlike guinea pigs, must synthesize and store the vitamin.	Injection of .01, .02, and .03 cc. solution yielded times of 3.5, 5, and 8 minutes respectively in rabbits. On the whole, the intradermal time was shorter in rabbits due to ability to synthesize the vitamin.
Cera (14)	Skin test is not a sensitive index of vitamin status.	Scorbutic animals yielded same scores as normal ones.
Rapaport and Miller (15)	The dye is chemically decolorized in the skin.	Equivalent amounts of injected methylene blue dye remained unchanged for at least 24 hours.
Masuzawa (16)	Times of less than 4 minutes denote saturation; above 10 minutes indicates deficiency state; 5-10 minutes is normal range.	There was close parallelism between changing weight and changing skin test time in normal and scorbutic animals.
Banerjee and Guha (17)	90 seconds is a minimal skin test value.	Guinea pigs on a supplemented diet revealed diminished times of 90 ± 5 seconds after 2-4 weeks.
Banerjee and Guha (18)	In the scorbutic group, the urine excretion and the plasma level is lower, and the intradermal time higher. However, within any group these relationships do not always prevail.	Variations of score within any one group did not allow accurate prediction of one score from another.
Banerjee and Guha (19)	Skin time is a good index of vitamin C saturation.	Maximal liver values of ascorbic acid were noted when the skin time became minimal (90 seconds).
Banerjee and Guha (20)	Disappearance of combined forms of urinary ascorbic acid is not related to tissue saturation.	Combined forms vanish prior to attainment of minimal skin times and maximal liver values.
Slobody (21)	Use a 4 mm. wheal and employ a dye concentration of N/300.	Larger wheals yielded most constant times. Higher dye concentrations minimized the personal equation by providing easier-to-read longer times.
Curatola (22)	The test defines only the extremes of ascorbic acid status of the skin.	Guinea pigs on a vitamin C-rich diet exhibited lower decolorization times than scorbutic ones.

A review of the literature on human studies[7] and an analysis of a group of our own patients will be considered in subsequent publications.

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LE TEST A ACIDO ASCORBIC INTRADERMAL PARTE I. UN REVISTA DE STUDIOS ANIMAL

Dr. med. e med. dent. E. Cheraskin, Dr. med. dent. J. B. Dunbar, e F. H. Flynn (B.S., M.T., A.S.C.P.)

SUMMARIO IN INTERLINGUA

Un revista del litteratura indica que omne le datos nunc disponibile es basate super le uso del colorante 2,6-dichlorophenolindophenol. Tabula 1 revela que (1) in omne studio, con tres exceptiones, le concentration del colorante esseva N/400, (2) in omne casos, con tres exceptiones, porcos de India esseva usate como animales experimental, (3) vibices, in tanto que reportate, habeva diametros de 2 mm, excepte in un caso, e (4) le quantitate del colorante usate in le experimentos esseva in omne casos, excepte in tres, 0,01 cm^3.

Tabula 2 summarisa le varie experimentos, le numero de animales usate e le stato de lor samitate, e le duration del experimentos.

Un examine de tabula 3 monstra que (1) solmente cinque del dece-quatro reportos offere le opportunitate de comparar le tempores de discoloration al initio e al fin del experimento, (2) le tempore del test intradermal es generalmente plus longe in animales scorbutic que in animales

normal, e (3) secundo le majoritate del investigatores il es un facto que le administration de acido ascorbic reduce le tempore de discoloration intradermal. Il es importante notar que in omne iste studios le accento esseva placiate super le mesurationes medie pro le gruppo integre plus tosto que super le variationes individual in le curso del experimento.

Le objectivos del studios animal, representate per tabula 4, pote esser summarisate como visante a (1) establir, in tanto que possibile, un correlation inter le tempore de discoloration intradermal e le concentration del vitamina in le histos, (2) determinar alterationes eventual in le tempore intradermal sub le effecto de supplementos de vitamina, (3) provar, si possibile, que le discoloration de 2,6-dichlorophenolindophenol reflecte un neutralisation del solution experimental plus tosto que un simple phenomeno de absorption, (4) correlationar le nivellos urinari e sanguinee de acido ascorbic con le tempores de discoloration, (5) comparar nivellos vitaminic del hepate con tempores del test dermatic, e (6) evalutar saturation corporee con acido ascorbic relative al disparition de formas combinate de acido ascorbic ab le urina.

Le resultatos obtenite es summarisate in tabula 5. Illos indica (1) que il existe un relation definite inter le nivellos del vitamina in le histos e le tempore intradermal, (2) que le tempore del test dermatic pote esser reducite per supplementation de acido ascorbic, (3) que 2,6-dichlorophenolindophenol es discolorate e non simplemente absorbite, (4) que gruppos de animales scorbutic e normal differe in lor excretion urinari de acido ascorbic, in lor nivello sanguinee del vitamina, e in lor tempore intradermal, (5) que maximos de saturation hepatic es correlationate con minimos de tempore dermatic, e (6) que il non existe un correlation inter le disparition de formas combinate de acido ascorbic ab le urina e le presentia de saturation in le histos.

Un revista del litteratura relative a studios in humanos e un analyse del observationes in un gruppo de nostre proprie patientes va esser le themas de publicationes futur.

Basic Science Reviews

J. Lewis Blass and

S. Sigmund Stahl, Editors

From time to time this section of the Journal of Dental Medicine will carry brief, but not necessarily definitive, reviews of basic science subjects pertaining to dental medicine. This material will be gathered specifically for the Journal. Clinical considerations, from the etiologic as well as the therapeutic view will be included. The following review by S. Sigmund Stahl is the first of this series.

It will be followed in future issues of the Journal by reviews on anatomy, bacteriology and metabolism of the periodontal structures in health and disease, studies in the pathology of various types of gingivitis, the effects of systemic disease and recent trends in therapy.

THE ROLE OF ANIMAL EXPERIMENTATION IN THE STUDY OF THE ETIOLOGY OF PERIODONTAL DISEASE

S. SIGMUND STAHL, D.D.S., M.S.*

One of the common meeting-grounds of medicine and dentistry can be found in the understanding of the effects of generalized disease states on the oral tissues as well as the effects of local dental disease on the overall health of the patient. This knowledge, though at present still limited, affects all fields of dentistry, since all tissues, whether calcified or soft, play a part in the maintenance of metabolic equilibria of the body. For the sake of brevity, this discussion will be limited to two problems namely, the effect of a local periodontal focus on general health and the effect of systemic disease on the periodontal lesion.

An answer to the first problem may be found in a recent comprehensive review article by Mitchell and Helman[1] who conclude that the periodontal lesion may become a significant etiologic factor in bacteremias, in the exacerbation of diseases such as subacute bacterial endocarditis as well as in some sensitization reactions. The opinions of these investigators is generally accepted by most students of the field.

The answer to the second problem still needs extensive investigation before any definitive answer can be given. Thus, the following thoughts are in many respects theoretical and require future research to prove or negate their validity. A common basis from which to proceed would be to accept periodontal disease as a syndrome complex with many local and general environmental factors playing a role in the clinical manifestations of the lesions (Figs. 1 & 2). The pertinent sections in any of the standard textbooks of periodontology give ample proof of the many contributing factors described by dental investigators, which influence periodontal pathology.[2, 3, 4, 5] In order to isolate and study the individual factors it is necessary to utilize animal experimentation

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Fig. 1: Clinical photograph of a 25-year old male diabetic, poorly controlled since childhood. Note the appearance of the gingiva.

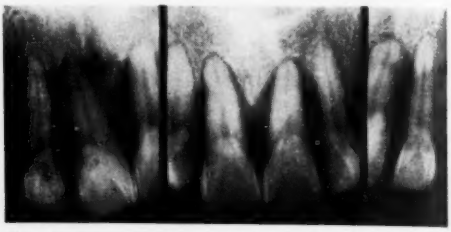

Fig. 2: Roentgenograms of the teeth and supporting alveolar bone of the case shown in fig. 1. Note the extreme resorption of the alveolar crest.

since in humans, combinations of all the factors commonly exists, thus making the study of any single unit virtually impossible.

Since most periodontists divide causes of periodontal disease into gingival irritation, occlusal trauma and systemic diseases, this paper will briefly review findings in experiments with animals when one or a combination of the above factors were used. It should be obvious that results from animal experimentation cannot immediately be transferred to human experience; however, certain basic mechanisms may be elucidated for further evaluation by clinical experience.

Gingival irritants, whether in the form of food impactions,[6] amalgam restorations made to irritate the gingivae[7] or interproximal wires[8] have been shown to cause gingival inflammation followed by recession and healing, if the tissue could withdraw from the irritant (Fig. 3). Occlusal trauma in the form of lateral or vertical stresses primarily cause changes in the peridontal membrane and alveolar bone in order to accommodate the abnormal occlusal stresses.[9, 10, 11, 12] However, trauma does not seem to affect the level of the epithelial attachment. Systemic debility whether in the form of nutritional deprivations,[13, 14] or severe systemic diseases, such as malignancies,[16] cause marked changes in the tooth attachment apparatus. These changes consist primarily of alveolar osteoporosis and a reduction in the number of young cells in the periodontal membrane. (Figs. 4 & 5). However, these deprivations acting alone do not seem to alter the level of the epithelial attachment. From these and other experimental studies, it would appear that both occlusal trauma and systemic deprivation do not initiate pocket formation at least at this level of research. Gingival irritation does produce injury of the epithelial attachment. However, unless the irritant is maintained and becomes progressively more severe, as is seen in food impaction for example, healing takes place with the epithelial attachment at a somewhat more apical level.

With these findings as basic considerations, one must then proceed to combine various of the above mentioned factors in order to study

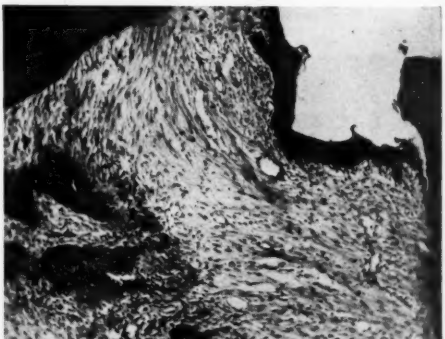

Fig. 3: Photomicrograph of an interdental papilla between the first and second molars of a rat carrying a wire irritant in the area over an eight-week period. Note the epithelial attachment slightly below the cemento-enamel junction and the marked osteogenic activity at the alveolar crest. Hematoxylin-eosin stain. magn. 100x.

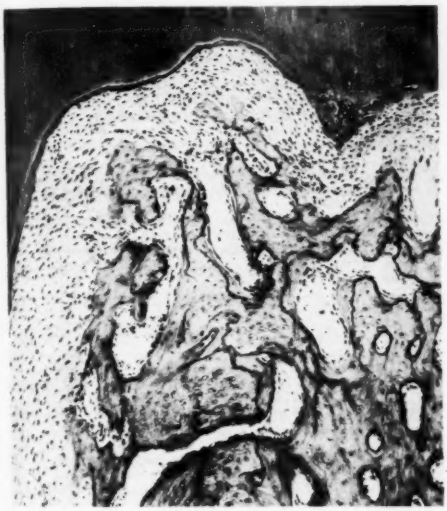

Fig. 4: Photomicrograph of the distal and crestal areas of the interradicular septum of the first molar of a rat, on a normal diet. Hematoxylin eosin stain. magn. 100x.

Fig. 5: Photomicrograph of the distal and crestal areas of the interradicular septum of the first molar of a rat on a protein-free diet for a six week period. Note the reduction in cellularity of the periodontal membrane and the enlarged marrow cavity as compared with similar structures shown in fig. 4.

the resultant effect of the combination on the periodontium of the experimental animal. When the effects of dietary deprivations are superimposed on locally irritated gingival tissues, one notes a retardation of repair which in turn results in a more profound injury.[17] (Fig. 6) Similar aggravation of the extent of injury by changing the local environment of the tissues might be produced by a combination of occlusal trauma and gingival irritation.[18] It thus appears that while gingival irritation initiates the injury to the epithelial attachment, changes in general as well as local environment of the tissues may influence the extent and severity of the resultant periodontal lesion.

The conclusion to be drawn from the above outline bears out the findings of Reynolds, who states, "when confronted with paradontal disease, one dentist or physician may feel that the solution to a particular problem lies in the field of endocrinology, another may feel that the dietary factor is the important cause. Still another may lean toward microbic retention as an explanation. The biochemist will undoubtedly lay most stress on plasma proteins, capillary

Fig. 6: Photomicrograph of an interdental papilla between the first and second molars of a rat on a protein free diet for eight weeks and carrying an interproximal wire irritant during the entire period. Note the presence of an inflammatory infiltrate below the epithelium as well as the position of the epithelium attachment in the apical third of the root. Hematoxylin-eosin stain. magn. 100x.

control, etc. The important point to remember is that the solution does not lie in any one field, the well-rounded physician and dentist will evaluate properly all possible approaches and attempt to place the patients' difficulty within its proper category.[19]

The clinical study of the patient includes[20] study of the occlusion, occlusal defects and irritational and systemic factors. "Further, local and general metabolic phenomena involved in periodontal pathosis are cared for by supportive therapy such as nutrition, medical diagnosis and treatment, uncovering of emotionally induced occlusal habits and other therapy included in the consideration of systemic factors."

It is in this spirit that the findings of the experimental approaches add to clinical observations so that together they contribute to ever improving therapy.

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Figures 3 and 6 appeared in Stahl, S. S., Sandler, H. and Cahn, L. R. The effects of protein deprivation upon the oral tissues of the rat and particularly upon periodontal structures under irritation. Oral Surg., Oral Med. and Oral Path. 8:760, 1955.

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LE ROLO DE EXPERIMENTOS ANIMAL IN LE STUDIO ETIOLOGIC DE MORBO PERIODONTAL

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SUMMARIO IN INTERLINGUA

Un del areas de interesse commun pro medicina e dentisteria es a trovar in le question del effectos de generalisate statos pathologic super le histos oral e del effectos de localisate morbos dental super le ben-esser general del patiente. Nostre cognoscentias in iste area, ben que al tempore presente illos es ancora limitate, concerne omne subdivisiones del dentisteria, proque omne histos—tanto calcificate como etiam molle—ha un rolo in mantener le equilibrios metabolic del corpore. In le interesse de brevitate, le presente discussion va esser restringite a duo

problemas specific: Le effectos de local focos periodontal super le stato de sanitate general e le effectos de morbos systemic super le lesion periodontal.

Proque le majoritate del periodontistas divide le causas de morbo periodontal in irritation gingival, trauma occlusal, e morbos systemic, iste articulo summarisa brevemente le constatationes obtenite in experimentos animal in que un del supra-mentionate factores o un combination de illos esseva introducite. On debe prender in consideration que resultatos ab experimentos animal non es immediatemente transferibile al situation human, sed super le base de tal experimentos il es possibile clarificar certe mechanismos fundamental usque a un puncto ubi illos pote esser evalutate plus detaliatemente per le experientia clinic.

Le conclusion derivabile ab le supra-delineate principios corrobora le constatation de Reynolds que "in le presentia de un caso de morbo paradontal, il es normal que un prime dentista o medico opina que le solution de un specific problema es a trovar in le campo del endocrinologia, durante que un secunde considera factores dietari como le causa principal." E Reynolds continua: "Un tertie dentista o medico prefere reguardar retention microbic como le melior explication. Le biochimico accentua sin dubita le question del proteinas de plasma, del regulation capillari, etc. Le facto principal, que on non debe unquam oblidar, es que le solution non se trova in ulle campo individual. Le medico e le dentista de cultura comprehensive es preste a evalutar correctemente omne le interpretationes possibile. Isto permitte a ille succeder in su effortio a effectuar le correcte categorisation del difficultate con que le patiente suffre.[19]"

NEW YORK SECTION

The New York Section of the American Academy of Dental Medicine elected new officers for 1957-58 last April. The following were unanimously elected:

President—Sanford Kirsch

President-Elect—Stephen Nachbar

Secy-Treasurer—Jack Theaman

Editor—Howard L. Ward

Delegate—Harold E. Brennan

Trustees—Theodore Lite, Seymour M. Koteen and Irving Yudkoff

A stimulating opening meeting is planned for October 16th. Dr. Saul Schluger will present an evening on "Newer Concepts of Periodontal Surgery." The presentation will consist of a projection and discussion of osteoplasty, osteoectomy and muco-gingival surgery.

The meeting will be held at 8 P.M. on October 16th, 1957 at the George Washington Hotel, 23rd Street and Lexington Avenue, New York City. A pre-meeting dinner is scheduled at 6:30 P.M.

HOWARD L. WARD

THE PATHOLOGY OF TRAUMATIC TEMPOROMANDIBULAR ARTHRITIS PRODUCED BY TRAUMATIC OCCLUSION*

DONALD A. KERR, D.D.S., M.S.†

The temporomandibular joint is a complex ginglymoarthrodial joint composed of three parts: the head of the mandibular condyle, the meniscus, and the articular eminence of the temporal bone. The meniscus is interposed between the head of the condyle and the articular eminence, and divides the joint into two parts. The periphery of the meniscus is attached on its borders to the capsular structures and muscle. This anatomical arrangement, in addition to partitioning the joint, produces two joint spaces: the condylar meniscal space between the condyle and the inferior surface of the meniscus, and the meniscotemporal space, which is between the meniscus and the temporal bone. Each joint space is lined by a synovial membrane which attaches to the border of the articular surface and the border of the meniscus. The inferior joint space (condylar-meniscal) permits movement of the head of the condyle in a sliding and rotary, or hinge, motion.

Before describing the changes produced by trauma, it would be well to review briefly the normal histology of the various parts of the joints.

The articular surface of the condyle is covered by a thin layer of fibrocartilage which is of fairly uniform thickness over the entire condylar head. The outermost portion is composed of dense collagenous fibers with few cellular elements. The fibrous layer on the surface serves as the perichondrium of the condylar head. The cartilage cells of the fibrocartilage increase in number as we progress toward the center of the condyle and soon blend into an area of hyaline cartilage. This zone of active cartilage formation is prominent during the period of growth, but gradually reduces in activity with advancing age; however, some proliferative activity can usually be observed. The cartilage gradually blends into trabecular bone which makes up the head of the condyle. In young individuals islands of cartilage may be observed in the trabeculae of bone; but this cartilage is replaced by bone in the adult individual. In older persons there may be a cortical plate of bone present beneath the thin layer of hyaline cartilage that separates it from the cancellous bone.

THE MENISCUS

The central portion of the meniscus is composed of a compact, dense, hyalinized, relatively acellular fibrous tissue. Near the borders of the meniscus the hyalinized fibers are less compact and the tissue is more cellular. At the periphery of the structure the tissue becomes more loosely arranged and blends into the capsule or muscle. The dense collagen fibers are arranged in an interlacing pattern presenting a felt-work appearance. The degree of hyalinization of fibers is not uniform and presents a mosaic pattern. Occasionally cells are interspersed between the collagenous fibers; some of these cells have a chondroid appearance and cause the area where the cells are present to resemble fibrocartilage. The central portion of the disc is avascular; in the periphery small vessels are present. The surface of the meniscus is covered by synovial cells. These cells are numerous and easily demonstrated near the periphery of the meniscus; near the center the cells are flattened and widely separated.

THE ARTICULAR EMINENCE

The surface of the articular eminence is covered by a layer of connective tissue, the outer portion of which is compact and relatively free of cells; the inner portion is more loosely

* Presented before the 13th Annual Meeting of the American Institute of Dental Medicine, November 5, 1956, Palm Springs, Calif.

† Professor of Oral Pathology and Periodontia, University of Michigan School of Dentistry.

arranged and more cellular. This surface does not have chondroid cells, and the connective tissue is in apposition to dense cortical bone. This layer serves as the periosteal covering of the trabecular eminence.

In function, the head of the condyle moves anteriorly and inferiorly along the articular eminence; the meniscus moves in the same manner so that it is always interposed between the two articular surfaces. At any point along the path of movement of the condylar head there may be a hinge movement. This movement takes place between the head of the condyle and the meniscus. It is during hinge axis movement that injury to the joint is most apt to occur, and the resultant changes will be most evident in the head of the condyle and the meniscus. When trauma is severe and results in displacement, injury may also be produced in the articular area of the temporal bone. For this reason the most severe changes associated with trauma are frequently seen in the condyle and the meniscus, especially when the injury is initiated by traumatic occlusion.

The changes produced in the temporomandibular joint due to trauma are inflammatory in nature. They vary in severity depending upon the degree and duration of the trauma. The greatest changes are observed in the articular surface of the condyle and in the meniscus. Acute changes are chiefly those of hyperemia and edema; but if prolonged or repeated, the changes become degenerative and proliferative. The character of the changes depends upon the portion of the joint involved. For ease of discussion, condylar and meniscal changes will be described separately.

MENISCAL CHANGES

The initial changes from injury of the meniscus are those of degeneration: there is hyalinoid and fibrinoid degeneration of the collagen fibers in the mid-portion, and there is some loss of fiber detail and tendency for myxomatous degeneration of the meniscus. The pattern of connective tissue fibers is interupted and clefts appear in the tissue. Some of the cells present become chondroid in character and small nodules of cartilage form in focal areas. There is an absence of inflammatory infiltration. On the surface of the meniscus there is an accumulation of fibrin in small amounts. These accumulations are usually associated with areas of change at the border of the meniscus. In longstanding injury there is vascularization of the areas of injury. Small vessels invade the areas of degeneration along with a proliferation of fibroblasts. After vascularization has occurred, inflammatory infiltration with a perivascular distribution takes place in the substance of the disc. In chronic cases there is a synovial cell proliferation which is most marked at periphery of the disc; here synovia is thrown up into villiform processes which are called synovial villi. Occasionally villi may be detached from the surface and undergo hyalinization and calcification. These free-floating, smooth, glistening, white bodies in the joint are called "joint mice." These bodies may be responsible for noise in the joint and also for locking of the joint. The accumulations of fibrin which are adherent to the surface of the meniscus become organized by proliferating synovial cells. The presence of the fibrin on the surface produces grossly a roughened appearance which may during function be responsible for the production of friction rub. The vessels of the border of the disc become sclerotic. In cases of great severity and long duration there is calcification of the areas which show vascularization. In very severe chronic cases there is cartilage formation which undergoes degeneration and calcification. There may also be evidence of ossification. This may be in the substance or on the periphery of the meniscus.

CONDYLAR CHANGES

The condylar changes most frequently observed are due to loss of elasticity of hyalin cartilage. The earlier changes are due to the loss of the fibrous tissue and fibrocartilage over the articular surface. The articular surface undergoes hyalinization, becoming entirely hyalin cartilage. Fissures and tears occur in the hyalin cartilage; some of the fissures and tears are of a horizontal nature and occur in the region of the chondral osseous junction. Resorption of bone occurs in this region and also prolifiera-

tion of fibrous tissue, especially in areas where the marrow spaces are open. With fibrous proliferation the area becomes vascularized. In these areas of vascularization and cartilage degeneration, calcification and ossification occur. This is especially true at the borders of the articular surface. These often extend for the full thickness of the articular cartilage and in some cases result in fragmentation of the cartilage. As the protective fibrous layer is destroyed, proliferation of cartilage occurs which increases the thickness of the articular surface. The proliferation is frequently rapid and focal and results in an atypical cellular cartilage. In some areas there may be extensive degeneration of cartilage which results in complete destruction and exposure of the condylar bone. The subcondylar cortex is destroyed and the marrow spaces exposed. The connective tissue of the marrow spaces proliferates and may produce cartilage between the osseous trabeculae. Complete destruction of cartilage on the articular surface results in eburnation of the condylar head.

At the periphery of the joint the cartilage is intimately connected with the joint capsule and the periosteum; proliferation of this peripheral area produces cartilage which becomes converted into bone by intermembranous bone formation. This proliferative activity produces alterations in the size and shape of the condylar head to form exostoses which are responsible for the characteristic lipping seen in traumatic arthritis.

Acute trauma of the temporomandibular joint produces pathologic changes that may allow luxation or subluxation of the joint and may permit the position of the joint to be altered. This altered position is frequently dictated by the occlusion and is frequently a traumatic position so that changes, sometimes marked pathologic changes, may be effective over long periods of time. The degree of pathologic change in the joint is dependent on the severity and nature of the trauma.

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LE PATHOLOGIA DEL TRAUMATIC ARTHRITIS TEMPOROMANDIBULAR PRODUCITE PER OCCLUSION TRAUMATIC

Dr. chir. dent. Donald A. Kerr (M.S.)

SUMMARIO IN INTERLINGUA

Le articulation temporomandibular es un complexe articulation ginglymo-arthrodial que consiste de tres partes: Le capite del condylo mandibular, le menisco, e le eminentia articular del osso temporal. Le menisco se trova interponite inter le capite del condylo e le eminentia articular e divide le articulation in duo partes. Le peripheria del menisco es attachate marginalmente al structuras capsular. Iste disposition anatomic resulta non solmente in le subpartition del articulation sed produce etiam duo spatios articular: Le spatio condylo-meniscal inter le condylo e le superficie inferior del menisco e le spatio menisco-temporal inter le menisco e le osso temporal. Ambe iste spatios articular es revestite de un meembrana synovial attachate al margines del superficie articular e del menisco. Le spatio inferior del articulation (i.e. le spatio condylo-meniscal) rende possibile que le capite del condylo executa movimentos glissante e rotatori.

Le alterationes effectuate per trauma in le articulation temporomandibular es de natura inflammatori. Illos varia in severitate secundo le grado e le duration del trauma. Le plus grande alterationes es observate in le superficie articular del condylo e in le menisco. Alterationes acute

es principalmente associate con hyperemia e edema. Quando illos es prolongate o repetite, tal alterationes deveni degeneratori e proliferative. Le character del alterationes depende del portion del articulation interessate.

Trauma acute del articulation temporomandibular produce alterationes pathologic que permitte luxation o subluxation del articulation o possibilemente alterationes in le position del articulation. Un tal alteration del position articular es frequentemente dictate per le occlusion. Illo es frequentemente un position traumatic, de maniera que le alterationes—a vices marcatemente pathologic—pote persister durante longe periodos de tempore. Le grado del alteration pathologic in le articulation depende del severitate e del character del trauma.

AMERICAN ACADEMY OF ORAL ROENTGENOLOGY

Scientific Session

Saturday, November 2, 1957, Hotel Lucerne

Miami Beach, Florida

9:00 A.M. "The Genetic Effects of X-radiation."
Berwind Kaufman, D.S., M.A., Ph.D., Research Investigator, Department of Genetics, Carnegie Institute of Washington.

10:00 A.M. "Recording Media and Viewing Techniques for Dental Radiography."
Dale Trout, B.S., D.Sc., Consulting Radiation Physicist, General Electric Company, Milwaukee, Wis.

12:00-2:00 Lunch.

2:00-2:15 "Report on Current Research in Dental Roentgenology."
Richard E. Richardson, D.D.S., Department of Oral Diagnosis and Treatment Planning, University of North Carolina School of Dentistry, Chapel Hill, N. C.

2:15-4:00 "Bone Architecture: Its Significance in Health and Disease."
Harry Sicher, M.D., D.Sc., Professor and Chairman of the Department of Anatomy and Histology, Loyola University, School of Dentistry, Chicago College of Dental Surgery, Chicago, Ill.

4:00-5:00 "Roentgenographic Interpretation of Periodontal Disease."
Beryl Ritchey, B.S., D.D.S., Past Secretary-Treasurer of the Colorado Dental Foundation, Colorado Springs, Colorado.

This is an open meeting and all who are interested are cordially invited to attend.

MECHANISM OF HEALING OF PERIODONTAL LESIONS*

HELMUT A. ZANDER, D.D.S., M.S.†

There is abundant clinical evidence that healing of periodontal lesions can and does occur.[1-4] This clinical evidence has been accepted by most workers in the field. However, there is as yet no unanimity on the mechanism or on the regularity with which such healing occurs.

In order to understand the mechanism of healing one must first take into consideration the normal attachment of the tooth to its surrounding tissues and, second, the mechanisms of healing and repair elsewhere in the body. Normally the epithelium which covers the gingival margin and lines the gingival sulcus terminates on the surface of the tooth and is then called the "epithelial attachment". This epithelial attachment is thought to be derived from the enamel epithelium; it consists of cells which are arranged more or less parallel to the surface of the tooth. With age, the epithelial cuff normally grows down onto the cementum. However, it is not clearly understood whether the degeneration of the periodontal membrane fibers, which are ordinarily attached to the cementum, cause this epithelial proliferation or whether the epithelial proliferation is responsible for the destruction of the fibers.

MECHANISM OF REATTACHMENT

Two studies by Waerhaug have shed considerable light upon the mechanism by which epithelial reattachment to the tooth surface occurs.[5, 6] In his first study the outer surface of the enamel was ground away well below the gingival margin with fine diamond stones. In this process the epithelial attachment obviously must have been removed. Moreover, the chances are that most of the epithelial lining of the gingival sulcus was injured or removed at the same time. Twenty-three days after removal and injury of the "epithelial attachment", it is seen that a new epithelial cuff has formed on the tooth surface. Thus it has become clear that if the original attachment produced by ameloblasts is injured either by mechanical trauma or by changes induced through calculus formation, healing with a new epithelial cuff can occur. Epithelium will not only reattach on tooth surface but also to crowns made of porcelain and acrylic, to acrylic root tips, and to other non-irritating materials. Reattachment of epithelium is not a new finding but something that has long been known to occur when teeth are transplanted or re-implanted, or after gingivectomy or a flap operation.

MAIN ATTACHMENT

The main attachment of teeth is provided by connective tissue fibers which are inserted in the cementum of the root on one side and the alveolar bone on the other. When pocket formation occurs, there is always loss of connective tissue fibers and downgrowth of epithelium. We recognize two types of pocket, based upon morphology.[7] "The gingival pocket, in which the bottom of the pocket is coronal to the level of the alveolar bone," and the infrabony pocket, in which the bottom of the pocket lies apically to the adjacent alveolar bone. The infrabony pocket seems to lend itself to a more favorable prognosis in regard to healing by connective tissue reattachment if treated properly and thoroughly. The basic microscopic features of such an infrabony periodontal pocket are: (1) The cementum is covered with calculus. (2) Between the calculus and the epithelial lining is an exudate full of inflammatory cells —very much the same as we discussed under the heading of "tissue reaction to calculus". (3) The adjacent epithelium is extremely thin —as a matter of fact, often absent in places. (4) Many new small capillaries lie close to

* Presented before the 13th Annual Meeting of the American Institute of Dental Medicine, November 6, 1956, Palm Springs, Calif.

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the surface of the soft tissue wall. (5) The connective tissue is almost devoid of its fibrous components and is loaded with round cells. These histopathological features explain the clinical symptoms of pus that can be pressed from such pockets and the bleeding which frequently occurs upon the slightest manipulation of the soft tissue. The question is how can healing occur in such a deep periodontal pocket?

TREATMENT

The treatment consists of instrumentation which implies removal of calculus and planing of the root surface and curettage. We previously discussed that therapeutic planing of the root surface also removed some or all of the cementum. This raised the question, whether this damages the tooth permanently. The only manner by which the injured cementum can heal is by new apposition of cementum, imbedding of new periodontal membrane fibers during this cementum formation, replacement of the chronically inflamed soft tissue with new connective tissue, and new bone formation. This healing is possible only through the process of creating a well organized blood clot and organization of granulation tissue. This is the process of healing anywhere in the body. Animal experimentation and studies on humans have shown that this process of healing also occurs in the area in which we are interested.[8, 9] While no hemorrhage can be expected from the tooth surface, the surrounding soft tissues bleed freely. If a blood clot is allowed to establish itself in this area, two things may interfere with its proper organization—infection or epithelial downgrowth. In animal experiments the clot is replaced by granulation tissue and within a month this granulation tissue has organized to form new cementum and bone, with a well-arranged new periodontal membrane. As a matter of fact, in studies which have had as their object the experimental production of periodontal pockets in animals one is impressed by the fact that it has been impossible to produce such pockets because the above described sequence of events has taken place in almost every case.

MECHANISM OF HEALING

The mechanism of healing in man is the same as in animals. In a specimen of an upper left cuspid which was obtained two months after pocket curettage one can see the same elements of healing. The roentgenogram, which was taken with a periodontal probe in place, shows a pocket 5 mm deep before treatment. Two months later we see the extent to which the cementum had been curetted away. On the surface of this cementum appears a pink-staining mass that may be called cementoid. It is lined on the soft tissue side with a row of cells that resemble cementoblasts. To this new hard structure connective tissue fibers are attached. In silver stained specimens these new connective tissue fibers cannot be distinguished from those attached to the uninjured cementum. New bone formation has occurred to bring the width of the periodontal membrane back to near normal. In other words, a mechanism exists by which healing of deep periodontal pockets in man can occur. The histological evidence corroborates the clinical findings.

IMPORTANT FACTORS IN TREATMENT

The factors that are important in successful periodontal treatment are: (1) Meticulous planing of the root surface. (2) Freeing of the soft tissue side of the pocket from epithelium. (3) Connection of all the walls of the pocket with a healthy blood clot. (4) Protection of the blood clot from mechanical injury and bacterial infection so that it can maintain its integrity. Therefore the operator should place over the curetted pocket a protective bactericidal pack to hold tissues, clot, and root surface in good apposition and protect the pocket from mechanical and bacterial injury. Unfortunately, at the present state of our knowledge we are unable to predict in every case whether or not the described mechanism of reattachment will occur. I am sure it depends upon proper selection of the case and the exactness of a meticulous technic.

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MECHANISMOS DE SANATION IN LESIONES PERIODONTAL

Dr. chir. dent. Helmut A. Zander (M.S.)

SUMMARIO IN INTERLINGUA

Le mechanismo sanative in humanos es le mesme como in animales. In un specimen de un dente canin supero-sinistre que esseva obtenite duo menses post le curettage de un bursa, on pote vider le mesme elementos sanative. Le roentgenogramma, que esseva effectuate con un sonda periodontal in sito, monstra un bursa de 5 mm de profunditate ante le tractamento. Duo menses plus tarde nos nota le grado a que le cemento esseva extirpate per le curettage. Al superficie de iste cemento un massa appare que produce un coloration rosa e que pote esser designate como cementoide. Al latere del histos molle, iste massa es revestite de un systema de cellulas que resimila cementoblastos. Attachate a iste nove structura dur on trova fibras de histo conjunctive. In specimens tractate per coloration a argento, iste nove fibras de histo conjunctive non es distinguibile ab illos que se trova attachate a cemento intacte. Ha occurrite un re-formation de osso que resulta in que le membrana periodontal reattinge un largor quasi normal. In altere parolas, il existe un mechanismo que es capace a effectuar le sanation de profunde bursas periodontal in humanos. Le studio histologic corrobora le constatationes clinic.

Le factores que es importante pro le successo del tractamento periodontal es le sequente: (1) Planage meticulose del superficie de radice. (2) Liberation del latere del bursa verso le histo molle ab epithelio. (3) Connexion de omne parietes del bursa con un ben-formate coagulo de sanguine. (4) Protection del coagulo contra lesiones mechanic e infectiones bacterial de maniera que illo pote mantener su integritate. Pro effectuar iste objectivo, il es importante placiar super le previemente curettate bursa un protective pacco bactericidial que mantene le histos, le coagulo, e le superficie de radice in le desirate apposition e protege le bursa contra lesiones mechanic e bacterial. Infelicemente, il es ver que le stato presente de nostre cognoscentias non permitte a nos predicer in omne caso individual si o non le supra-describite mechanismo de re-attachamento va functionar. On pote esser certe, nonobstante, que le successo depende del correcte selection del casos e del exacte observation de un technica meticulose.

Medical Views on Oral Problems

TREATMENT OF PENICILLIN REACTIONS

Pronestyl is not the final answer to treatment of penicilin reactions, but . . . [it is] at least as good as and probably better than Benadryl for this purpose. In severe reactions cortisone or ACTH may be indicated, especially in light of reports of anaphylaxis and death from severe penicillin reactions. Such cases are unusual, however, and it is felt that these powerful metabolic drugs are unnecessary in average cases. Pronestyl will afford rapid relief of symptoms in many of these cases. . . . [It] is relatively nontoxic, although . . . one case of allergy to this drug itself has been reported. Some care must be taken in patients with heart disease because of the possibility of the development of heart block. We noted no untoward effects in our patients except for mild transient dizziness in one patient, anorexia in another, and insomnia in a third. Electrocardiograms taken after two or three weeks on Pronestyl therapy showed no significant abnormalities, and there was no evidence of agranulocytosis or other hematologic disorder. It is . . . felt that this drug has a place in the treatment of allergic reactions to penicillin and deserves further clinical evaluation.—P. B. Jennings, M.D., and S. Olansky, M.D., The Use of Procaine Amide in the Treatment and Prevention of Penicillin Reactions, *Annals of Internal Medicine, April,* 1954.

THE BACTERIOLOGIC STATUS OF THE PULP CHAMBERS IN INTACT TEETH FOUND TO BE NONVITAL FOLLOWING TRAUMA†*

The contents of the pulp chambers of 46 teeth of children and young adults were examined bacteriologically after a blow sustained by these teeth. The teeth were not fractured but were traumatically devitalized as evidenced by discoloration or failure to respond to thermal stimulation, electrical pulp testing, or both. Aerobic and anaerobic cultures were made. Of the 46 teeth, 38 yielded bacterial growth. The number of bacterial strains recovered from an individual tooth varied from none to 6. A total of 71 strains were isolated. The largest single group consisted of aerobic streptococci (20 strains). Next in order were micrococci (15 strains). Anaerobic cocci constituted the third largest group (12 strains). Twenty-three of the 71 strains were anaerobes. These findings show that bacteria can be recovered from the pulp chambers of most traumatically devitalized teeth even though there is no direct communication between the pulp and the oral cavity. It is postulated that most of these organisms reached the pulp from the gingival sulcus by way of the lymphatics and blood vessels of the periodontium.

† By J. B. Macdonald, G. C. Hare and A. W. S. Wood. Oral Surg. 10:318-322 (March) 1957 [St. Louis].
* Medical Literature Abstracts, J.A.M.A., June 22, 1957, p. 935-936.

MUCOSAL CARCINOMAS AS A RESULT OF IRRADIATION†*

The authors present 9 patients in whom carcinoma arose in mucous membranes included in a field of radiation therapy. In 6 of these patients squamous cell carcinomas of mucous membranes developed in areas treated by external roentgen irradiation from 9 to 25 years before appearance of the tumors. Each patient experienced late radiodermatitis in the skin utilized for the external port. There was also fibrosis of the tissues between the skin and the affected mucosa. There was microscopic evidence of radiation change in the mucosa and submucosa of all the cancer-bearing areas. Treatment was unsuccessful, and the authors believe that this failure was due to the multicentric origin of the tumors, with new foci developing in the irradiated mucosa at a rate impossible to control. Three of these tumors, the 2 of the tongue and the 1 of the anus, developed in patients treated for cancer of adjacent organs or tissues. The anal lesion could hardly be a late localized recurrence of the cancer of the cervix treated 11 years before; the one epidermoid carcinoma of the tongue could not be a recurrence of malignant mixed tumor of the parotid, which showed no local persistence after 9 years. The other tongue lesion occurred in an irradiated area 13 years after successful radiotherapy for a presumed cancer of the nasopharynx. The 2 hypopharyngeal cancers occurred in patients treated for inflammatory cervical lesions, one of them tuberculous adenitis. The squamous cell cancer of the buccal mucosa represents the culmination of a compound tragedy of errors in a patient whose ossifying fibroma of the upper maxilla was erroneously diagnosed as adamantinoma and treated with irradiation over a period of years. Even if the tumor had been an adamantinoma the irradiation was incorrect treatment.

In 3 patients adenocarcinoma developed. An adenocarcinoma of the transverse colon developed directly beneath an obvious area of skin change on the anterior abdominal wall where irradiation had been used as treatment for a duodenal ulcer four years before. The time interval is short as compared with the intervals in reports of other irradiation-induced cancers, but the previous treatment and residual

objective findings at least suggest a causal connection. The carcinoma of the rectosigmoid occurred in the field of irradiation in a woman treated for cancer of the uterine cervix. Again the time interval is only three years, but there is demonstrable irradiation change in the intestinal wall about the tumor. In the patient with the adenocarcinoma of the cervical esophagus the long interval between irradiation and tumor formation (25 years) and the obvious irradiation changes in the tissues of the neck indicate that this is almost certainly an irradiation-induced cancer. The epithelium of mucous membranes, like that of the skin, is capable of malignant tumor formation as a late effect of irradiation.

† By D. P. Slaughter and H. W. Southwick. A. M. A. Arch. Surg. 74:420-429 (March) 1957 [Chicago].
* Medical Literature Abstracts, J.A.M.A., June 1, 1957, p. 606.

CHRONIC LYMPHOCYTIC LEUKEMIA IN A FAMILY INCLUDING TWIN BROTHERS AND A SON*

This is an unusual article describing a case of chronic Lymphocytic leukemia in twin brothers. The exceptional interest is that not only was there leukemia in twin brothers, but an identical type of leukemia which later developed at approximately the same age in the son of one of the twins. Additional reports of identical twins having the same type of leukemia are also reported.

* By Frederick Gunz, M.D. and William Dameshek, M.D. Abstracted by Leonard M. Nevins from J.A.M.A., July 20, 1957. pps. 1323-1325.

BAD BREATH*

TO THE EDITOR:—*A 20-year-old girl has had bad breath for some years. She has excellent oral hygiene and no sinus disease, and the tonsils have been removed. Her dietary habits are good, and her past health has been excellent. What metabolic or other studies might be made in an attempt to arrive at a diagnosis?*

M.D., Kentucky.

ANSWER.—This is an extremely common complaint. Unpleasant breath is difficult to control in many individuals. There are many causes for bad breath. They have to do with the type of bacterial flora found in the mouth as well as in the digestive tract. Constipation, or at least unsatisfactory bowel evacuation, is frequently a factor. Gases absorbed from the intestinal tract and breathed through the lungs are the chief origin of unpleasant breath. Its control is often extremely difficult. It may require brushing of the teeth many times a day, and chewing gum and carrying lozenges in the mouth are helpful. Preparations of chlorophyll are helpful, but this must be in adequate concentration. Half a teaspoon of powdered charcoal, placed on the back of the tongue 10 or 15 minutes before meals and taken with water, is very effective in controlling gas accumulations in the digestive tract and has helped many. There is no one very effective way of controlling this unpleasant condition.

* Queries and Minor Notes, J.A.M.A., June 15, 1957, p. 827.

SIMPLE METHOD FOR PROTECTING FRESH SMEARS FROM DRYING AND DETERIORATION DURING MAILING*

As exfoliative cytology has become more widely used the need for an adequate technique for mailing fresh, unstained smears to a cytology laboratory without exposing them to drying, which is known to distort the structural characteristics of the cells and their nuclei, has been more keenly felt.

The method presented in this report provides a preliminary fixation of the smears and the formation of a film which protects the smear surface during mailing. The staining reaction of the cells shows no appreciable change.

Fresh, wet smears are covered immediately with a small quantity—approximately 0.25 or .5 cc per smear of Diaphane (a synthetic resin made by the Will Corporation, Rochester, N.Y.) solution (3 parts of 95% ethyl alcohol to 2 parts of Diaphane). After the Diaphane has dried thoroughly, in 20 to 30 minutes, a hard, smooth film protects the surface of the smear. The slides can then be wrapped in wax paper and mailed any distance. When received in the laboratory, they are placed in alcohol-ether for approximately 20 minutes to remove the Diaphane; then they are stained according to the routine method for cytological smears.

The fixation, preservation, and staining of all types of cells in smears from respiratory and female genital secretions were found to be very satisfactory even after a lapse of two weeks. Previous use of the method has shown that the staining of the cells remains unaltered for several years.

The adoption of this technique could be expected to help in extending the use of the cytological method of diagnosis by making it available to areas not yet serviced by a local laboratory as well as to a larger group of private physicians, and dentists.

* By George N. Papanicolaou, M.D., Ph.D. and Emma Lou Bridges, New York. Abstracted by Leonard M. Nevins, from A.M.A., July 20, 1957, pps. 1330-1331.

ESSENTIAL BILATERAL NEURALGIAS OF THE TRIGEMINAL NERVE: IN CONNECTION WITH 13 PATIENTS 6 OF WHOM UNDERWENT OPERATION†*

A review of records of more than 500 patients with essential facial neuralgia treated by one of the authors disclosed 13 instances in which the condition was bilateral. The true incidence of bilateral

neuralgia, however, is higher than that indicated by these figures, because many of the 500 patients were lost to follow-up. Those with an adequate follow-up (1 year or more) comprised nearly 200 who were treated by neurotomy and about 30 who were treated medically, making the proportion of those in whom both sides of the face were affected 5 or 6%. Facial neuralgia is rarely bilateral at the outset. Only 2 of these patients had pain on both sides of the face when they were first seen. The choice of therapy lay between an operation on the side of the face more severely affected and bilateral tractotomy. The unilateral procedure seemed preferable because of the definite asymmetry of the pain in these patients. The 1st, a 62-year-old woman, refused operation and was lost to follow-up. The 2nd, a 50-year-old woman, was first treated by neurotomy on the left side, on which the pain was predominant. Complete relief of pain on both sides followed the operation and lasted for 8 years. Pain then recurred on the right side of the face and was eventually relieved by neurotomy on that side. The treatment must be regarded as a success, because even though the patient when last seen had occasional, not very severe attacks on the left, she had been free from pain on that side for 17 years after the first operation.

The other 11 patients had contralateral pain beginning at various intervals after the initial operation. Six were treated medically with alcohol infiltration, administration of vitamin B_{12}, and other measures. The remaining 5 underwent total radicotomy carried out by the usual technique, with the temporal approach and preservation of the motor root, which must be identified and scrupulously respected. The results were excellent: not only was the pain relieved but there was no facial paralysis, no ocular complications, and especially no late difficulty in chewing, although a transient difficulty followed the operation in some cases. The value of retrogasserian radicotomy carried out by the Spiller-Frazier technique is fully vindicated by the findings in these patients, in 3 of whom the results have remained excellent for more than 8 years. Bulbospinal tractotomy may be indicated in a young person with true bilateral facial neuralgia, but in the usual form of the disease, in which involvement of the 2nd side occurs long after that of the 1st, the Spiller-Frazier operation produces a satisfactory and lasting functional result.

† By P. Wertheimer and J. Descotes, Presse méd. 65: 265-267 (Feb. 13) 1957 (In French) [Paris].
* Medical Literature Abstracts, J.A.M.A., June 15, 1957, p. 818.

BURNING AND BAD TASTES IN THE MOUTH*

Commonly, in older women and occasionally in older men, there comes a miserable burning in the tongue or mouth, perhaps on only one side. There may also be a foul or metallic taste. When the trouble is only on one side the physician can be pretty sure that it is due to injury in a nerve center. I have seen this syndrome occur so often after definite strokes that now, when I see a person with it and cannot get a history of a stroke, I always suspect that one came during the night. In these cases I have never seen any treatment do any good, and the burning sensation may remain for years.

* From "Little Strokes" by Alvarez, J.A.M.A., April 2, 1955, p. 1202.

WHITE BLOOD CELL COUNT AND HYPOTHYROIDISM*

TO THE EDITOR:—*In general, is there an increase in the white blood cell count of patients with hypothyroidism?*

Ursula G. Mandel, M.D., Los Angeles.

ANSWER.—No change in the white blood cell count of patients with hypothyroidism has been reported. Of course, anemia is frequently encountered in patients with myxedema; this anemia does not respond to therapy with liver and iron but does respond when the myxedema is properly treated with thyroid hormone.

* Queries and Minor Notes, J.A.M.A., Aug. 10, 1957, p. 1727.

DRYNESS OF THE MUCOUS MEMBRANES*

At the meeting of the Belgian Gastroenterological Society in February, Weill reported a series of seven patients (six women and one man) with dryness of the mucous membranes (Sjögren's syndrome). This syndrome is characterized by deficiency of tears associated with secondary lesions of the conjunctiva and cornea, deficiency of saliva, dryness in the nose and throat associated with a typical cough, and, if the syndrome is in an advanced stage, hoarseness. Microscopic examination reveals atrophy of the secretory glands. Deficiency of secretion (achylia) was also found in the stomach and pancreas. A decrease of the intestinal secretion is manifested by constipation, perianal pruritus, and congestion of the hemorrhoidal vessels. The syndrome is most frequently associated with rheumatic arthroses. The cutaneous symptoms include dry skin, brittle hair, and keratosis around the hair follicles. The disease occurs chiefly in women and seems to become manifest in the menopausal period. All the patients in this series had had dryness of the mucous membranes of long duration. The author pointed out a new symptom, typical of this disease: the characteristic aspect of the rectal mucosa covered with patches of adherent feces. This was observed only when there was no previous preparation or enema. The treatment is symptomatic and includes cervical sympathectomy, parasympathomimetics, electrocoagulation of the out-

lets of the lacrimal canaliculi, artificial tears, and various vitamins, but the results are disappointing. The author, however, obtained excellent results with a combination of larger doses of vitamin A and oxapropanium iodide. Under its action perspiration, defecation, and lacrimation were restored.

* Foreign Letters, J.A.M.A., Aug. 10, 1957, p. 1699.

BONE LESIONS IN LEUKEMIA*

At the meeting of the Belgian Radiological Society in March, Haesendonck called attention to two types of bone lesions encountered in the roentgenograms of children with leukemia. There are either destructive lesions (such as generalized osteoporosis, osteolytic foci, and metaphysial lesions) or productive lesions (such as the foci of osteosclerosis or periosteal reactions). These lesions are not modifications of leukemia but rather the expression of a disturbance of the ossification mechanism due to the infiltration of pathological leukocytes into the bone marrow. These changes are mostly found in the long bones. The transverse osteoporotic bands, localized in the metaphysical area, previously attributed to other causes, are frequently seen. Their extent progresses with the course of the disease but seems to be reversible. Of 39 children, 15 showed bony lesions and were retained in the hospital.

* Foreign Letters, J.A.M.A., Aug. 10, 1957, p. 1699.

ADDICTION TO MEPROBAMATE (EQUANIL, MILTOWN)*

To the Editor:—Evidence is steadily accumulating that abrupt withdrawal of meprobamate from patients who have been ingesting this drug in large amounts may be followed by major convulsions. Barsa and Kline (*Am. J. Psychiat.* 112:1023 [June] 1956) observed single convulsions in 6 to 25 schizophrenics who had received 2.4 Gm. of meprobamate daily for six months; Lemere (*J. A. M. A.* 160:1431 [April 21] 1956 and *A. M. A. Arch. Neurol & Psychiat.* 76:205 [Aug.] 1956) noted a seizure in one patient who had taken 6.4 Gm. of meprobamate daily for a month; and Tucker and Wilensky (*Am. J. Psychiat.* 113:698 [Feb.] 1957) reported convulsions in 2 of 32 psychotic patients who had received 1,600 mg. of meprobamate, increasing to 4,800 mg. daily, for three months. Both Lemere, and Tucker and Wilensky, also remarked on postwithdrawal anxiety and tension in a significant percentage of cases. Recently, Swinyard and others (*Science* 125:739 [April 19] 1957) reported hyperexcitability and decrease in the threshold for electrically induced seizures in mice chronically treated with meprobamate in doses increasing from 300 mg. to 1,800 mg. daily for eight days.

Observations made at the U. S. Public Health Service Hospital, Lexington, Ky., confirm these reports. After abrupt withdrawal of meprobamate, one female patient who had been ingesting 3.2 Gm. of meprobamate daily complained of anxiety, insomnia, excess salivation, and headaches. Although a convulsion did not occur, abnormal electroencephalograms, showing paroxysms of mixed fast and slow activity, were observed 29 to 77 hours after the last dose of meprobamate. A male patient who had been taking 4 Gm. of meprobamate daily for about three months had nervousness, headache, and one grand mal convulsion after discontinuation of use of meprobamate. No definite evidence of abstinence was observed after withdrawal of the drug from two patients who had been taking only 1.6 Gm. daily. Gradual withdrawal at a rate of 0.8 Gm. daily from a male patient who had been receiving 3.2 Gm. daily was followed by insomnia and tremulousness but no convulsions.

Five dogs were given 3.2 Gm., increasing to 8.2-8.8 Gm. of meprobamate daily for four to six months. One animal died after two months before withdrawal was attempted. After abrupt discontinuation of the drug all of the four remaining dogs showed marked restlessness, tremors, violent motor hyperactivity, and frequent licking movements. All four dogs had convulsions, and three died after three to five seizures.

These data show unequivocally that continued ingestion of large dosages of meprobamate can create physical dependence, manifested on abrupt withdrawal of the drug by hyperirritability of the central nervous system and convulsions. The type of addiction caused by meprobamate resembles that caused by chronic intoxication with excessive amounts of barbiturates or alcohol. As is true of barbiturates, addiction to meprobamate will probably occur in only a very small proportion of persons who receive the drug therapeutically. Nevertheless, care should be exercised in prescribing meprobamate. Patients receiving meprobamate should be supervised closely and immediate steps taken if their consumption of the drug begins to increase. Especial care should be exercised in prescribing meprobamate for alcoholics or narcotic drug addicts. Prescriptions for meprobamate should not be refilled. The drug should be withdrawn slowly, rather than abruptly, from patients who have been ingesting it chronically.

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* Correspondence, J.A.M.A., July 20, 1957, p. 1382.

AGRANULOCYTOSIS AFTER TRANQUILIZER*

An inquest was held after a 43-year-old woman had died of agranulocytosis after treatment in a men-

tal hospital with pacatal (Pecazine), a tranquilizer. The coroner stated that this drug, freely obtained from drugstores, could damage the bone marrow. In his opinion it should be given only to patients who are psychotic, and only when other forms of treatment have failed. A pathologist testified that blood tests made during treatment gave a false sense of security, since effects on the bone marrow could appear suddenly. Because so little is known about the drug, it should not be used in general practice. The dosage given to this patient was not excessive and there was no reflection on the medical staff.

* Foreign Letters, J.A.M.A., Aug. 3, 1957, p. 1608.

EXCESSIVE X-RAY THERAPY TO THE SKIN*

TO THE EDITOR:—*What are the prophylaxis and treatment in the case of a patient who received too large a dosage of x-ray therapy for a seborrheic type of facial dermatitis four years ago?*

M. A. Wuerschmidt, M.D., Oswego, N. Y.

ANSWER.—The question is very vague. There is not enough information given regarding the degree of radiodermatitis, nor is there any information given regarding the dosage of x-rays received to the face. This is important because, if the x-ray therapy was given four years ago and the dosage was in excess of what the skin could tolerate, then by this time there should be definite radiation changes. The amount of radiation changes depends upon the amount of radiation which was received four years ago. The treatment therefore depends upon the amount of radiation damage which is present now. From the query it is assumed that the radiation changes are minimal at the present time; otherwise, the word "prophylaxis" would not have been used. If the radiation changes are minimal, then, of course, the best thing to do to prevent further damage to the irradiated skin is to protect the skin in every way possible by applying emollients at night; these emollients include cold cream or petrolatum or even ointments containing both vitamins A and D. There is no specific topical remedy that can be applied to the face which would cure radiodermatitis or which would prevent radiodermatitis if a dosage of radiation is given in excess of what the skin can tolerate.

In addition to using emollients, the patient who has been irradiated should avoid exposure to ultraviolet rays, both natural and artificial. Sun bathing is bad for such an individual. If he does go out in the sun, then he should apply a sun-protective preparation; there are many such "sun screeners" on the market. It is preferable that a greasy sun screener be applied rather than a sun screener whose base is greaseless. Exposure to extremes of temperature should be avoided. Going out in the cold wind tends to make a person who has been irradiated have a tender skin. The cold wind might even cause fissures to occur on such a skin. Of course, such a person should not have any further radiation. Even the taking of dental films should be kept down to an absolute minimum. If the radiation damage is severe, plastic surgery and skin grafting may have to be resorted to. Perhaps the simplest solution to a problem like this is to obtain the advice of a local dermatologist.

* Queries and Minor Notes, J.A.M.A., Aug. 3, 1957, p. 1628-29.

POTENTIAL HAZARDS OF MEPROBAMATE (EQUANIL, MILTOWN)*

Since its introduction almost two years ago, the tranquilizing agent meprobamate has been received with considerable enthusiasm by the general public as well as by large segments of the medical profession. This widespread interest has been based primarily on the assumption that large doses of the drug can be administered with practically no side effects. It has become increasingly apparent that meprobamate is capable of producing a rather wide variety of side effects and untoward reactions.

Hypersensitivity reactions have occurred with sufficient frequency to indicate that these are definite and not infrequent complications of meprobamate therapy. Dermal manifestations have included urticaria and diffuse maculopapular and erythematous skin rash, often accompanied by intense pruritus. Hypersensitivity reactions of this type have occurred in patients with no previous history of allergy or prior exposure to meprobamate.

Acute nonthrombocytopenic purpura is another potentially serious type of sensitivity reaction to meprobamate. Symptoms have included widespread purpuric, erythamatous, petechial dermatitis, peripheral edema and finer phatelit counts are not affected, but there is some evidence of increased capillary permeability.

No definite antidote or regimen of therapy for over dosage has been devised. It is therefore, important that meprobamate be prescribed with considerable discretion and in small quantities, if at all, in patients who may have suicidal tendencies.

There is also some evidence that meprobamate possesses habit-forming properties. Withdrawal symptoms, including convulsions in some patients, have been observed when use of the drug has been discontinued abruptly after prolonged therapy.

In addition to physical dependence, psychic dependence with a tendency toward excessive self-medication is undoubtedly created in certain patients.

This report points out that side-effects and untoward reactions to meprobamate can and do occur and that the drug should be administered with the same discretion as other therapeutic agents.

* Report of Council on Drugs. Abstracted by Leonard M. Nevins, from J.A.M.A., July 20, 1957, pgs. 1332-1333.

Book Reviews

Oral Medicine, by Lester W. Burket, D.D.S., M.D., 558 pages, 391 illustrations including a color atlas of 10 plates, 3rd ed., J. B. Lippincott & Co., Philadelphia, $14.00.

The first two editions of this book enjoyed widespread use and this third edition seems destined for the same success.

For those practitioners who are interested in the oral cavity, dentist or physician, this is a most valuable book. It is comprehensive and almost all inclusive. It would appear that in a text of this magnitude, there should have been included a section on the findings in the normal mouth.

The book is well written. It is divided into twelve sections, each section subdivided into specific categories. Local oral disturbances are treated in separate sections from those of a systemic nature.

Each disease entity, when possible, is considered with a brief description of the condition, and there are headings in bold type to denote sections on symptoms, treatment, prognosis and oral aspects. This enables the reader to quickly pick out the material he wishes to find.

There is an extensive index at the end of each section for those who wish more information on a subject. The mechanics of the book are good, the illustrations and color plates well reproduced.

The book should be read by every person in the field of oral medicine. The student and practitioner will find this an excellent reference book to have available at all times. Burket's Oral Medicine is recommended to all.

LOUIS R. BURMAN

Dental Clinics of North America, a symposium on: 1. New Developments in Operative Dentistry by Drexell A. Boyd, D.D.S. 2. Differential Diagnosis of Prosthodontic Needs by Raymond J. Nagle, D.M.D. W. B. Saunders Co., Philadelphia, 1957.

With the publication of this first issue of Dental Clinics of North America, the publishers are fulfilling a long-standing need in the profession. The tremendously rapid accumulation of new procedures, concepts and technics have made it increasingly more difficult for the busy practitioner to keep pace with all of the current developments. By combining the efforts of outstanding clinicians and teachers in the form of symposia on a particular subject, the publishers are able to present the last word on the specialty under discussion.

This first issue is devoted to two fields—Operative Dentistry and Prosthodontics. The editors are well-renowned and the contributors represent a Who's Who in their respective specialties.

Of particular interest in Part 1 are three very timely articles on high speed preparations and instrumentation, a presentation on the rubber-base impression technics and a well written summarization of endodontic principles.

The second part, which deals with the prosthodontic needs of the patient, contains in addition to the technical contributions two very desirable papers on the esthetic considerations involved in denture construction.

If this first vloume is any portent of the future contents of the Dental Clinics, then these publications will certainly become standard reference texts and a necessary part of every dentist's library.

ALEXANDER SOBERMAN

Clinical Pedodontics by Sidney B. Finn, D.M.D., M.S., and E. Cheraskin, M.A., M.D., D.M.D.; Joseph F. Volker, M.S., D.D.S., Ph.D.; H. Perry Hitchcock, D.M.D., Joseph P. Lazansky, M.D., D.D.S., D.M.D., Gilbert J. Parfitt, F.D.S., M.R.C.S., L.R.C.P., D.M.D., Aceeb E. Thomas, D.M.D., and John J. Sharry, D.M.D., of the University of Alabama School of Dentistry. W. B. Saunders Co., Philadelphia. 664 pages, 275 figures, 1957. $12.00.

This fine textbook on dentistry for children has been prepared for the teaching of undergraduate dental students and for the general practitioner. The eight authors are all members of the faculty of the University of Alabama School of Dentistry.

As dentistry progresses, greater emphasis is being placed upon prevention in order to maintain an ideal oral structure. The authors have devoted a large portion of this textbook towards this end. They have tried to make the chapters on preventive orthodontics and the prevention of dental caries unique in clarity and comprehensiveness. Among the subjects which have been included in this textbook are parent counseling, the problem of pain, oral diagnostic procedures, antimicrobial agents, extensive pulpal techniques, the handling of the handicapped child in the dental office, the construction of mouth protectors, the role of heredity, biopsy techniques and hospital admittance procedures.

The text is well written, well illustrated, and the type is large and clear which makes for easy reading. There is a list of references at the conclusion of each chapter which will be of great aid to the reader who is interested in pursuing this subject further.

The eight authors have shared the editorial burden of the 28 chapters in this text. Although the topics vary in length and scope, the text is well integrated and there is a continuity of subject throughout. This book should be in the library of every practitioner, whether he emphasizes pedodontics or not.

IRVING YUDKOFF

Biochemical Disorders in Human Disease, Editors: R. H. S. Thompson and E. J. King, Academic Press, Inc., New York, $12.50.

Our newer knowledge of the process underlying disease now takes us beyond the limits of classical cellular pathology into the realms of abnormalities resulting from disorders of the enzyme catalyzed reactions occurring within the cells. The pathology of many diseases is now passing from the stage of morbid histology to that of morbid biochemistry. The rapid strides in biochemical theory and technique is made possible by the penetration of cellular mechanisms in disease enabling the biochemical interpretation of disease processes especially abnormalities of enzyme action and deviations in electrolyte balance. This book contains the contributions of outstanding scientists on the mechanisms of biochemical disorders in human disease. Many of the revelations not only elucidate pathogenesis but clarify diagnoses and management of both medical and surgical patients. The chapters are given in terms of organ basis rather than in terms of biochemical concepts. Each chapter thus presents the various diseases of any organ systems featuring on biochemical disorders and advancements. The approach is individual but the context is clear and concise. Many biochemical disorders have been omitted i.e., of the skin, infectious diseases and cancer because of the heterogeneity of the biochemical advances and the difficulty of their correlation for clinical application. The book is a veritable mine of recent advances in biochemistry of human disease.

I. NEWTON KUGELMASS, M.D.

Partial Dentures, A Practical Textbook, by Kenneth P. Austin, D.D.S., F.I.C.D. and Ernest F. Lidge, Jr., D.D.S., B.S., C. V. Mosby Company, St. Louis, 1957, 227 pages, 165 illustrations, $9.50.

This is an excellent practical and psychologic approach to partial prosthesis construction for both the practitioner and beginner in dentistry. It encompasses not only many clear, well done illustrations and captions for practical usage in all phases of partial prosthesis but the correct dentist-patient attitude towards these restorations.

It is cleverly divided into five convenient sections for the reader. The first section is devoted to construction of the partial prosthesis from the time of the first examination of the patient to the time when laboratory procedures are required.

The second section covers the laboratory procedures which are very instructive for the beginner and might suggest additional ideas to the practitioner. Part three continues from the point at which the denture is completed by the laboratory to completion of the case. This section combines proper methods of checking peripheries, framework, esthetics and function with patient education for partial prosthesis.

The fourth section is devoted to the student having to do all of his own laboratory work and an assist to the dentist doing immediate prothesis.

Section five clearly outlines the many pitfalls that are involved in the construction of a partial prothesis. It suggests ways of anticipating and eliminating in a concise manner many of these problems.

The print is large and clear which makes for easy reading. The lack of a complete bibliography is a little disappointing in an excellent book of this type.

LEONARD M. NEVINS

Pediatric Dentistry, by M. Michael Cohen, D.M.D., and 16 collaborators, 607 pages with 395 illustrations, C. V. Mosby Co., St. Louis, $14.75.

The dental practitioner who is interested in children will be interested in reading this book. The editor and his collaborators have treated the child as a growing entity and this is an important facet in the treatment of the normal and the handicapped child, both types being covered in the text.

Seventeen authors have contributed to this book, each in his own particular field. Development of the child is covered in all phases and various methods of treatment are indicated, not alone in the operative field but in the surgical and systemic areas.

The book is well organized and well written. The mechanics of the text are good. Bold type on coated stock makes for easier reading. The illustrations are well done and are nicely reproduced. For the general practitioner and the student, this book is recommended reading.

LOUIS R. BURMAN

Oral Hygiene, by Russell W. Bunting, D.D.S., D.D.Sc., D.Sc., and collaborators, 3rd. Edition, Lea & Febiger, Philadelphia, 334 pages. 233 illustrations and 1 plate in color. $7.00.

This text is directed specifically to the needs of a dental hygienist and serves as an excellent text for them. Much of the material which has appeared in former editions has been revised and rewritten. Two of the contributors are members of the dental hygiene profession.

This enlarged edition contains a new chapter on the pathology of dental caries. The chapter on pre-

vention has been enlarged to include a comprehensive outline of dietary regulations for the control of dental caries. New chapters on the role of the dental hygienist in public health, and on the history and organization of dental hygiene, have been contributed by dental hygienists. All other chapters have been revised and amplified to make them useful to teachers and students of dental hygiene.

Much of the material on preventative dentistry and all of the techniques on oral prophylaxis are equally adaptable to teaching of dental students and graduate dentists. The text contains a great deal of factual data, concisely stated, and of special interest to dental hygienists.

This enlarged third edition is a welcome addition to the dental literature.

Psychopharmacology, published by American Association for the Advancement of Science, edited by Nathan S. Kline, 1956. 175 pages, 11 tables, 3 figures, $3.50.

This monograph embodies some major contributions on the new pharmaceuticals in the field of mental disease. Kinross-Wright maintains that we have no panacea in chlorpromazine although an extremely valuable compound which contributes much to the treatment plan but does not suffice by itself. Hollister contends that tranquilizers produce considerable improvement but it is not the beginning of the end of therapy but rather the end of the beginning. Other workers are more cautious about dosage and effects alone or in combination with each other or with other drugs on the central sympathetic and automatic nervous systems. Each contribution is followed by discussion of the participants of the Conference, hence the monograph is invaluable for psychiatrists in private and hospital practice.

I. NEWTON KUGELMASS, M.D.

Dental Health Education, by Frances A. Stoll, Ed. D., R.D.H., 196 pages, 16 illustrations, Lea and Febiger, Philadelphia, 1957, $4.50.

This text is primarily designed for those interested in setting up dental health programs in schools, communities and organizations.

The book is divided into three sections. Part I considers the foundations for dental health education, and other settings in which dental health education functions. Part II discusses the various methods of dental health education in schools. Part III takes up audio-visual aids, radio and motion picture instruction, lectures, discussions and other instructional materials. The final section, and a very short one, presents opportunities available for patient instruction in the dental office. Chapter 2 in Part I really has no place in the book and should have been omitted. It deals with diseases and infections of the mouth and the reader should be referred to scientific books on these topics rather than to try to use it as it has been presented in this volume.

The book is well written and many useful and practical areas of endeavor are covered. It is a practical guide to planning for dissemination of dental health information to children of school age. For those interested in this phase of activity in the dental field, this is a worthwhile book.

LOUIS R. BURMAN

The Dental Business Office, by C. Edward Rutledge, D.D.S., and Edward H. Winsor, Lea & Febiger, Philadelphia, 152 pages, 1956, $3.50.

Here is a practical guide for every progressive dentist. The primary purpose of every dentist is to render a health service. He must provide for his family, meet business expenses and realize the best possible return from his practice. To help every modern dentist reach these goals is the purpose of this text.

A dentist and a dental business manager present to the reader their own ethical, highly successful methods for improving office efficiency and securing greater personal income. The authors recognize the fact that dentists, no matter how deeply concerned with the professional aspects of dentistry, realize that proper office management is essential to a successful practice.

The need for proper financial arrangements and how to make them are considered early in the book. This is followed by a description of several accepted credit policies. The profitable recall service is considered and a successful, inexpensive system thus has proved more effective than the usual card mailing is described.

Dentists and senior students will find in this helpful book a solution to their problems on the practice management side of dentistry.

Clinical Dental Roentgenology, by John Oppie McCall, D.D.S., F.A.C.D., and Samuel Stanley Wald, D.D.S., F.A.C.D., ed.4., W. B. Saunders Co., Philadelphia, 466 pages, 1445 illustrations on 529 figures, 1957, $10.00.

This new fourth edition is a remarkably practical book encompassing and tying up all loose ends of every phase of dental roentgenology. The text contains detailed and lavishly illustrated step-by-step techniques for both long cone and standard intraoral pictures. There is a detailed description and evaluation of the latest equipment including high voltage apparatus and ultra-speed films plus the most effective darkroom techniques. Of particular interest is the current data on radiation hazards to both dentist and patient. How to calculate safe exposure limits is thoroughly discussed. The interpretation of findings

is handled for every dental disorder demonstrable on the roentgenogram. Differential diagnosis is emphasized.

Of unique value to specialists is the authoritative coverage of conditions in Orthodontia, Pedodontia, Periodontia, Oral Surgery, Oral Diagnosis, etc. Seven chapters deal directly with Pedodontia. Periodontal disease is fully covered from the roentgenologic standpoint. Prognosis as indicated in the roentgenogram is clearly presented. The subject of oral diagnosis is presented from the standpoint of the interpretation of the roentgenogram, including roentgenographic requirements for interpretation.

The printing and illustrations are clear and distinct. The meticulous index permits one to locate any particular problem in a matter of moments. The dental practitioner will find here a comprehensive and richly illustrated guide to all phases of dental roentgenology.

Local Anesthesia and Pain Control in Dental Practice, by Leonard M. Mondheim, B.S., M.S., D.D.S., C. V. Mosby Co., St. Louis, 1957, Price $8.75.

Dr. Monheim, a recognized authority on dental anesthesia, has presented an excellent text devoted to the over-all control of pain in dental practice.

Special commendation should be accorded T. W. Brand, D.D.S. for a comprehensive and interesting discussion of the trigeminal nerve.

Techniques of regional anesthesia and analgesia are clearly and concisely explained. They are complemented by outstanding photographs and illustrations which permit the reader to follow the procedures very readily.

In the chapter devoted to local anesthetics and anesthetic solutions most of the proprietary drugs are discussed to include their chemistry and pharmacological action. Of particular interest are the personal experiences of the author during the use of various anesthetic agents.

Anesthetic complications and office emergencies are explored, together with the procedures to prevent and properly handle these unfortunate incidents.

This text stresses the need for careful planning for the control of pre-anesthetic anxiety and postoperative pain. Apprehensive patients are fearful of the experience to come and find it difficult to control their emotions. Also, patients are often permitted to leave the dental office totally unprepared for any discomfort that may follow dental procedures. A prescription for some medication will give them a sense of security and assurance during the post operative period.

All practitioners can benefit by the many suggestions drawn from the author's vast experience in clinical anesthesia and in the control of pain in dental practice.

MARVIN N. OKUN

Interviewing, Counseling and Managing Dental Patients, by Joseph Bregstein, D.D.S., Prentice Hall, Englewood Cliffs, N. J., 510 pages, 1957.

In presenting this text to the dental profession, Dr. Bregstein has demonstrated effectively that technical skill is not sufficient to provide a successful practice. He stresses the need for knowledge of the psychological management of the patients in the dental office, in order to gain their good will and cooperation.

The author outlines certain techniques to be employed in solving basic problems in every practice. There are simple methods described for coping with objections raised by patients regarding pain associated with dental procedures.

In order to establish effective patient-dentist relationship, the author maintains that a demonstration of professional skill be given to the patient. This can be accomplished by citing cases of similar complexity which were successfully treated. Constant patient education throughout the entire series of appointments is necessary in order to maintain patient enthusiasm and interest.

Other portions of the text are devoted to a discussion of the use of music in an office, handling of young patients and dental psychiatry and hypnosis.

Both the experienced practitioner and the fledgling can use this text as an adjunct in the management of the dental patient.

Review of Dentistry, edited by James T. Ginn, D.D.S., Ed. 2, C. V. Mosby Co., St. Louis, 997 pages, 1956, $8.00.

This text, now in its second edition, has three basic purposes. The first is to serve as a concise, comprehensive and authoritative guide for those preparing for State and National Board examinations. The second is to serve as a general review for the undergraduate student, while the third purpose is to serve as a handbook and a ready reference book for the busy general practitioner. Dr. Ginn and his twenty six consultants have fulfilled these three basic purposes more than adequately.

There are twenty-two chapters of which about seven deal with the basic sciences. The remaining fifteen chapters cover the clinical phases of dentistry including subjects on nutrition, jurisprudence and practice management and ethics.

The answers to questions are given as briefly as is compatible with clearness, with a minimum of explanation. For a more detailed coverage on a particular subject, the student is referred to general textbooks.

A text of this type is an asset to the dental student, the practitioner and to the postgraduate student who is preparing for certification examinations.

Practical Dental Monographs, Technic for Difficult Lower Impressions, by Ralph C. Appleby, D.D.S., Year Book Publishers Inc., Chicago, 29 pages, 14 figures, published bi-monthly, annual subscription $7.50.

This short monograph is an excellent review on the technic for difficult lower impressions. In these 29 pages the general practitioner will find a discussion of anatomy, impression technique, pressure versus non-pressure technique and full coverage and extension versus minimum extension techniques. The illustrations are clear and self-explanatory. An extensive bibliography refers the reader to other authors who deal with the same problem.

These bi-monthly monographs will deal with a different subject every issue and should be of great aid to the practitioner with a specific problem.

A Manual of Stomatology for Dental and Medical Practitioners, by J. P. Walsh, M.B., B.S., D.D.Sc. (Melb.), L.D.S. (Vic.), F.D.S.R.C.S. (Eng. and Edin.) M.D.S. (N.U.I.) Hon. Causa., N. M. Peryer Ltd., Christchurch, New Zealand, 130 pages, 37 illustrations, 1957, New Zealand price 30/-.

This book is based on a course of ten lectures given to fifth year medical students at the University of Otago. Brevity and broad principles rather than details are discussed in this text. Each chapter could be readily developed into a book. The dental student and practitioner should find this text a useful summary of an extensive field. The general medical practitioner will find here the minimum knowledge necessary for a better understanding of the dental and oral problems he is likely to meet in his practice.

Although this text is modest in size and deliberately brief in style, it covers a wide range of subjects which are the mutual concern of the medical and dental practitioners. It is not only of practical value to medical and dental students, both undergraduates and post graduates, but also contributes to a better understanding and closer collaboration between the two professions.

Foreign Correspondents Report

The Journal of Dental Medicine now has seventeen Foreign Correspondents in sixteen foreign countries. Their responsibilities to this Journal include the reporting of the progress of dentistry and dental medicine in their respective countries. In addition, they are constantly looking for articles of merit by the leading dentists in their own countries. These articles will be translated into English and forwarded to the editor for publication in our Journal.

Our correspondent in Rio de Janeiro, Dr. Jacob Reifman reports that Dr. Samuel Charles Miller, Founder and First President of the American Academy of Dental Medicine "presented lectures before the Brazilian Dental Association and the State Hospital. He also participated in the meeting of Associacao Paulista de Cirurgioes Dentistas in Sao Paulo. After leaving Sao Paulo, Dr. Miller visited and lectured in Montevideo, Buenos Aires and Lima."

Our foreign correspondent in Burma, Dr. Wu Moon Aung, writes us that the President of the Union of Burma "conferred an unprecedented honour on the dentists in Burma when he asked Dr. Moon Aung to present to him all the Founders and Office-bearers of the Burma Dental Association."

Left: A group at the Associaçao Brasileira de Odontologia: From left to right: Dr. Jacob Reifman, Chairman of Foreign Relations Committee; Dr. Samuel Charles Miller; Dr. Wladimir de Souza Pereira, President of Uniao Odontológica Brasileira; Dr. Evandro de Castro, President of Asociaçao Brasileira de Odontologia; and Dr. Orandino Prado Filho, President of ARPA Brasileira.

Right: Dr. Samuel Charles Miller and Dr. J. Reifman, who was the Chairman of the session at the Associaçao Brasileira de Odontologia in Rio de Janeiro. The subject of Dr. Miller's lecture was "Prevention and Treatment of Periodontal Disease."

"An unprecedented honour was given to the Dentists in Burma when Dr. Moon Aung was asked by H. E., The President of the Union of Burma, to present to him the Founders and Office-bearers of the Burma Dental Asociation in the Throne-room of the President's House." (L. to R. standing) A. D. C., Dr. Teo Eng Hwa, Dr. D. S. Ahluwalia, Dr. R. L. Soni, Dr. S. S. Mathur, Dr. M. A. Nakhuda, Dr. J. M. Shaw, Dr. Than Aung, Dr. Thein Aung, A.D.C. (L. to R. sitting) Dr. Thomas Minn, Dr. William Tan, Dr. Wu Moon Aung, "Mr. President", U Win Maung, Dr. Kyee Myint, Dr. G. V. Thakur, Dr. G. D. Kapur.

Academy Proceedings

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Twelfth Mid-Annual Meeting and Luncheon at the Hotel New Yorker, New York City, on Sunday, December 8, 1957. There will be a business meeting for members at 10:00 A.M. This will be followed by a luncheon and scientific session for Academy members and friends starting at 12:30 P.M.

The afternoon scientific session will be devoted to a symposium on "Rehabilitation." Participating in this symposium will be:

Arthur J. Barsky, M.D.; New York, N. Y.
Attending Plastic Surgeon, Albert Einstein College of Medicine.

TOPIC—"Rehabilitation Through Plastic Surgery."

Robin M. Rankow, M.D.; New York, N. Y.
Adjunct Surgeon, Montefiore Hospital; Assisting Attending Surgeon, Columbia Presbyterian Hospital; Assistant Professor, Columbia University; Consultant Editor, Head and Neck Cancer, New York State Dental Journal.

TOPIC—"Immediate and Delayed Rehabilitation of the Head and Neck Cancer Patient."

Howard A. Rusk, M.D.; New York, N. Y.
Professor and Chairman, Department of Physical Medicine and Rehabilitation, New York University Bellevue Medical Center; Associate Editor, *The New York Times*.

All members and interested dentists and physicians are cordially invited to attend. For reservations and programs address the Chairman, Dr. Sheldon J. Ross, 136 East 36th Street, New York, N. Y., or Dr. Andrew Cannistraci, 2152 Muliner Avenue, The Bronx, New York.

Twelfth Mid-Annual Meeting Committee
Dr. Sheldon J. Ross, *Chairman;* Dr. Nathan Wachtel, *Co-Chairman;* Dr. Andrew Cannistraci, Dr. George Goldberg, Dr. Rudy Palermo, Dr. Abraham M. Reiner, Dr. Stephen Nachbar, Dr. Irving Yudkoff.

Twelfth Annual Meeting

AMERICAN ACADEMY OF DENTAL MEDICINE

Montreal, Canada

May 29, 30 and 31, 1958

DR. SAMUEL CHARLES MILLER TO BE HONORED

The Periodontia and Oral Medicine Department of New York University College of Dentistry will tender a testimonial dinner to Dr. Samuel Charles Miller, Professor and Chairman, on Saturday evening, Dec. 7, 1957 at the Hotel Roosevelt, Madison Avenue and 45th Street in New York City. Dr. Miller will be honored for the completion of twenty-five years as Chairman of the Department of Periodontia and Oral Medicine.

Reservations should be made as soon as possible since there will be a limited attendance. Dress is optional. Ladies are cordially invited. The dinner will be $15.00 per person. Please make checks payable to Dr. George J. Witkin, Treasurer, 501 Madison Avenue, New York 22, N. Y.

SIDNEY SORRIN, *Chairman*

DEAN HINE RECEIVES TUFTS UNIVERSITY AWARD FOR LEADERSHIP

Dr. Maynard K. Hine, dean of Indiana University School of Dentistry received the Tufts University School of Dental Medicine Award for "Leadership in Periodontology." The presentation was made last June at the Eighth Annual Berkshire Conference in Oral Pathology and Periodontology conducted by Tufts University at Lenox, Mass. (L. to R.) Dr. Cyril D. Marshall-Day, Tufts dean, Dr. Hine, and Dr. Irving Glickman, Director of Postgraduate Studies at Tufts University School of Dental Medicine.

The citation reads as follows: "In recognition of a distinguished career as a teacher of Periodontology and in tribute to your life long devotion in pioneering effort to make the benefits of the science of Periodontology available for the service of mankind, Tufts University School of Dental Medicine is pleased to confer upon you this Award for Leadership in Periodontology."

Among his other accomplishments, Dr. Hine is editor of the *Journal of Periodontology* and co-author of three books on periodontology and preventative dentistry.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Samuel Charles Miller and Staff:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE

Ten sessions, Wednesdays, 4:00 to 6:00 P.M. February 12, 1958 to April 23, 1958.
Tuition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE

Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
March 3 to March 28, 1958 inclusive
Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 16, 1957 to May 28, 1958.
Tuition $1,010.00. Instruments and books approximately $135.-00.

No. 122 B. Two academic years, full time. September 16, 1957 to May 27, 1959.
Tuition $1,770.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 16, 1957 to May 27, 1959. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 16, 1957, to May 26, 1961. (Two and one half days weekly.)
Tuition $1,770.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

No. 222. GRADUATE COURSE IN PERIODONTIA AND ORAL MEDICINE

Two academic years, full or half time, beginning Sept. 16, 1957. Tuition $2,020.00 (two years). Instruments and books approximately $275.00.

This program leads to the degree of Master of Science (in Dentistry). The objectives are to teach superior proficiency in Periodontia and Oral Medicine based on adequate background in the basic sciences.

ENDODONTICS COURSE N.Y.U. COLLEGE OF DENTISTRY

The New York University College of Dentistry announces a forty hour postgraduate course in the Principles and Practice of Endodontics beginning Wednesday, October 23, 1957. The class will meet Wednesdays and Fridays, 9:00 a.m. to 1:00 p.m. for five weeks. The principal objective will be to teach practical methods of treating pulp-involved teeth to maintain them in a healthful, functioning condition.

Emphasis will be on the newer developments in Endodontics and advances in the field relating to Endodontics. Lectures, demonstrations and clinical practice will constitute the course.

Dr. Morris B. Auerbach, Associate Professor and Chairman of the Department of Endodontics, and members of the Endodontics Department will teach this course.

Tuition: $200.00
Instruments: $65.00 (approximately)
The class enrollment is limited to ten members.

Further information and applications may be secured by writing: Director, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N. Y.

BETH ISRAEL HOSPITAL, BOSTON, MASS.

The Dental Department of the Beth Israel Hospital announces a postgraduate course in Periodontia to be given in the Fall of 1957.

A two week seminar in Periodontia is scheduled for October 7 to October 18. The objective of this seminar is to present to the practitioner all of the techniques employed in periodontal therapy as well as theoretical background. It will include etiology, diagnosis and treatment of various diseases of the supporting tissues of the teeth. Periodontal pocket therapy as well as an over-all mouth rehabilitation program will be stressed. Local environmental factors and systemic influences in periodontal therapy will be discussed. The material will be presented by lectures, demonstrations, and clinics. The tuition fee is $350.00.

Co-Directors of the Course are Henry M. Goldman, D.M.D. and Bernard Chaikin, D.M.D.

For further information, write to: Director of Public Relations and Education, Beth Israel Hospital, 330 Brookline Avenue, Boston 15, Massachusetts.

UNIVERSITY OF CALIFORNIA ESTABLISHES RESEARCH-TEACHER TRAINING CENTER

The School of Dentistry, University of California in San Francisco has instituted a Research-Teacher Training program under the direction of Dr. Hermann Becks, Professor of Dental Medicine. The U.S. Public Health Service in Washington has awarded the School a Graduate Training Grant for this purpose for a period of five years.

The Center will provide facilities, and instruction in basic research and the principles of biology as they pertain to Dentistry. Each year three or more trainees will be placed on a concentrated schedule, under the guidance of the Program Director and associates, to teach first some of the fundamentals of animal experimentation, various types of operations, autopsies, etc. This will be followed by intensive schooling in methods of tissue preparation, and a complete study of the gross and histologic results. In conjunction with this basic training, the scholars may enroll in courses of general and oral histology and pathology for a better understanding of tissue reactions as influenced by systemic processes. It also will provide a good background for a teaching career and advanced scientific investigation as well as clinic practice. If the trainee is qualified and wishes to do so, he may take work leading to the M.S. and/or Ph.D. degrees. The annual stipend ranges from $4,000 to $6,000.

Further information may be obtained by writing to Dr. Hermann Becks, School of Dentistry, University of California, San Francisco 22.

AMERICAN ACADEMY OF PERIODONTOLOGY

The American Academy of Periodontology will hold its Annual Meeting at the Hotel Di Lido, Collins Ave. at Lincoln Rd., Miami Beach, Florida, on Oct. 31st, Nov. 1st and 2nd, 1957.

This meeting is open to all members of the American Dental Association upon the payment of a modest fee.

This year's meeting will emphasize latest advances in research and clinical study of periodontal involvements.

HOWARD A. HARTMAN, D.D.S.,
Chairman; Publicity Committee,
American Academy of Periodontology.

TEMPLE UNIVERSITY SCHOOL OF DENTISTRY

Clinical Endodontics: October 14, 15, 16, 17, 18, 19, 1957 by Leonard Parris, D.D.S.—Assistant Professor of Endodontics, Temple; and Herbert Schilder, D.D.S.—Chief of Endodontics, Beth Israel Hospital, Boston; Instructor in Endodontics, Tufts Dental College; and associates.

The objective of this course is to train the participant in clinical endodontic procedures. Sufficient material of a basic biologic nature will be presented to insure a proper understanding of the clinical phase.

The course will consist of lectures, supplemented by visual material and clinical demonstrations. The postgraduate student will utilize diagnostic procedures, initiate treatment, and carry through to completion endodontic therapy on clinic patients provided. Single and multi-rooted teeth will be treated. Surgical therapy will be demonstrated.

Fee: $150.00. Class limited to six (6).

Five half-days will be spent by the participants treating clinic patients. This course can be used as the prerequisite for the participation course in **Immediate Root Resection,** scheduled for June 2-7, 1958.

For additional information and application, write to Dr. Louis Herman, Postgraduate Division, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

THE UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

37 refresher courses will be offered by the University of Alabama School of Dentistry in Birmingham, Alabama during the 1957-58 academic year. These are listed below according to the months in which they will be given:

SEPTEMBER: Office Accounting and Income Tax Guidance for the General Practitioner, English for Professional People, The Use of Drugs in Dental Practice, Practical Root Canal Therapy.

OCTOBER: Partial Denture Prosthesis; Office Management and Psychology of Dentist-Patient Relationship; Insurance, Investments, and Estate Planning; Oral Pathology-Oral Medicine; Dental Materials, Practical Application and Evaluation of Newer Advances and Changing Concepts; Effective Speaking for Professional People.

NOVEMBER: Oral Rehabilitation, The Theory and Practice of Periodontics, Histopathology of the Periodontium, Dental Assisting, Emergency Care in Dental Practice.

DECEMBER: Laboratory Techniques, Practical X-ray Technique and Interpretation.

JANUARY: High Speed Techniques in Operative Dentistry, Treatment Planning and Case Presentation, Immediate Root Canal Therapy Followed by Apicoectomy, The Management of Pain in Dental Practice.

FEBRUARY: Nutrition and Dentistry, Effective Speaking for Professional People (eight evenings during the month), English for Professional People (16 one evening per week sessions), Periodontology for the General Practitioner, Preventive Orthodontics, Laboratory Procedures in Crown and Bridge Prosthesis for the General Practitioner.

MARCH: Occlusal Adjustments; Insurance, Investments, and Estate Planning; Income Tax Guidance; Applied Maxillofacial Anatomy; Practical Exodontia.

APRIL: Root Canal Therapy, Pedodontics for the General Practitioner.

MAY: Complete Denture Prosthesis, Management of Infections in Dental Disease, Surgical Anatomy of the Head and Neck followed by Etiology and Diagnosis of Pain of Dental Origin, and Removable Orthodontic Appliance Construction.

Additional information about these refresher courses can be obtained by writing Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham 3, Alabama. Information about graduate and postgraduate programs is available through the office of the Dean.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces two Research Fellowships in Oral Pathology and Periodontology.

These fellowships intended for graduate dentists interested in research and teaching careers in oral pathology and periodontology are now available. In addition to participation in an extensive research program, the fellowships offer training in oral pathology in a laboratory with an active biopsy diagnostic service, and an opportunity for preparing for advanced degrees. There is an annual stipend of $5000 and the fellowships are renewable.

Additional information may be obtained from Dr. Irving Glickman, Department of Oral Pathology and Periodontology, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Mass.

GREATER NEW YORK DENTAL MEETING TO FEATURE "TODAY'S EMERGENCIES"

The 33rd annual Greater New York Dental Meeting, world's largest and most comprehensive seminar of postgraduate dental education, will be held at Hotel Statler, New York City, December 9 to 13, 1957.

This year's program will cover the latest findings and developments in all phases of general and specialized practice, including the newest in equipment and materials.

A special program portion devoted to "Today's Emergencies" will present four essayists who will cover systemic involvement, endodontics, periodontics and surgery.

A session on complete mouth rehabilitation will offer a functional and dynamic approach to the problems of vertical dimension, centric relation and centric occlusion. Techniques utilized in treating extensive rehabilitation cases will be described and demonstrated.

Use of new impression materials, new denture bases and the most modern concepts of esthetics, covering complete service from examination of the patient through installation of partial and full restorations will be analyzed and explained.

Other program highlights will include the role of stress in dentistry, effects of high speed instrumentation on the pulp, geriatrics, and a wide range of other topics.

Further information will be furnished promptly on request to Mrs. Mabel Purdy, Executive Secretary, Room 106A Hotel Statler, New York 1, N. Y.

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